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TEXAS STATE JOURNAL OF MEDICINE

Volume 46 Number 9 Austin, Texas, September, 1950

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President's Page

DOCTORS FACE SERIOUS WARTIME RESPONSIBILITIES

The repercussions of war, the magnitude of which cannot be determined at this time, are beginning to be felt very definitely by doctors. Regardless of the policies which have been pursued and circumstances which have come about as a result of Washington's policies in the last few years, the facts at this moment (August 29) are that the only doctors the Army can call by present law are a comparatively few reserve medical officers.

It now seems it will be only a few days until doctors in great numbers are going to be made available to the military services by the enactment of legislation in Washington. Such legislation has been advocated by the American Medical Association for a long time.

The Executive Council of the State Medical Association of Texas on August 24 approved legislation endorsed by the American Medical Association, and apparently this legislation will be endorsed by the majority of the doctors. The details will not be discussed here.

I feel that all doctors must continue to show a fair and understanding attitude toward each other and to the medical profession as a whole as it attempts to meet its obligation under war circumstances.

It is clear that if we doctors cannot meet our major responsibilities in time of war, we may expect to lose the confidence of the public in us to solve medical problems in normal and peaceful periods.

It is not quite clear yet what the repercussions of this war will be on the fight that has been made by American medicine against socialization of our profession. Nor is it clear what the effect of this war is going to be upon the move toward general socialization. I am of the opinion our struggle is going to be made harder rather than easier. This means each doctor, in the service and out, must continue to watch the future of medicine very carefully.

Of particular interest in our fight against socialization of medicine is the attitude of the Texas Pharmaceutical Association. Under the leadership of its secretary, Mr. C. J. M. Roesch, this organization is undertaking to carry forward an education program against socialized medicine. This program will culminate in November with an intensive drive in which the Junior Chamber of Commerce will lend full co-operation to the Texas Pharmaceutical Association and its secretary, Mr. Roesch.

Our public relations staff endorses this program highly. It is my opinion county medical societies will want to cooperate with the Texas Pharmaceutical Association and Mr. Roesch as they bring this program to your communities. I am sure you will be hearing about it.

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DOCTOR DRAFT LAW PASSED

Legislation providing for the drafting of physicians, dentists, and other specialists according to a priority system approved by the American Medical Association was passed by both houses of Congress and signed by President Harry Truman shortly before this JOURNAL went to press. Major provisions of the bill are as follows:

1. Physicians and dentists (and other specialists who may be named by the President) who have not reached the age of 50 and are not members of military reserves must register by a deadline to be set by the President.

2. Any registrant is subject to induction if acceptable to the military, but the following priority is specified:

a. Former ASTP and V-12 men who have not served on active duty and others deferred from service to continue their education during World War II and who have had less than ninety days of active duty.

b. Members in the above group who have had more than ninety days of active duty but less than twenty-one months.

c. With no reference to the above groups, those who did not have active service subsequent to September 16, 1940, including postwar medical graduates or other physicians who have not served and are not yet 51 years of age.

d. All others, including World War II nonreserve veterans. Men in this group will be called on the basis of extent of duty—those with the least duty first.

3. After registration, the Selective Service will assign each registrant to one of the priority categories. If he is called for service, the registrant will be ordered to take a physical examination and then will have twenty-one days to set his affairs in order before reporting to the induction station for commissioning and assignment.

4. A registrant may apply for a reserve commission. If he is accepted, he will be subject to military orders through the reserve service and will not be processed further by Selective Service. Physicians now in the reserve forces are already subject to military orders and do not come under the provisions of this act.

5. Every reserve officer called to duty, on a

voluntary or involuntary basis, is eligible to receive a $100 bonus per month. Men required to register under this act may qualify for the $100, but only if they volunteer prior to actual induction.

6. Deferment is at the discretion of local Selective Service boards; however, the President is authorized to provide for certain deferments. Action will be based on the registrant's previous military service, his dependency status, and undue hardship that might ensue. National, state, and local advisory committees are charged also to give consideration to civilian as well as military needs and the "maintenance of national health, safety, or interest" in granting deferments. A national advisory committee "which shall advise the Selective Service system and coordinate the work of state and local volunteer advisory committees with respect to selection of needed" professional personnel is provided for by the act; the committee must include representatives of the medical and dental professions.

The act states that "it is the sense of Congress" that Selective Service deferments will be granted to premedical, predental, and allied students in numbers equal to the present enrollment.

7. Inductees under the act must serve twenty-one months.

8. This act is an amendment to the Selective Service Act and will expire with that law next July unless extended by Congress.

Dr. R. A. Trumbull, Dallas, chairman of the Council on National Emergency Service, expected to call a meeting of the council September 17 in Austin to formulate plans for activating county procurement committees to function in accord with the new law. Most county medical societies already have procurement committees appointed and standing by.

It is anticipated that the draft law will be in operation soon and that it will permit a more orderly and fairer induction of physicians into active service than has been possible in the past few months. Members of these advisory committees and every physician affected, whether he is called to service himself or must carry a greater civilian load because his colleagues enter the armed forces, should look at the situation objectively and attempt to do his share in seeing that adequate medical care is available to soldier and civilian alike.

AMERICAN MEDICINE ADVERTISES

Almost every literate American will have an opportunity during October to read the message of the American Medical Association that freedom of enterprise, especially in the field of medicine and health insurance, is best. In October the American Medical Association will have paid advertising in 11,000 daily and weekly newspapers—all of the bona fide English language papers in the United States, Alaska, and Hawaii; in approximately 30 of the leading national magazines; and on approximately 300 radio stations throughout the country. These messages will be carried to readers and listeners regardless of the editorial policy of the paper or station concerned.

In many instances newspapers are issuing special medical supplements in conjunction with the advertisement, thus giving other organizations, institutions, and business firms an opportunity to publish "tie-in" advertisements and providing space for news and feature articles on medical and health subjects of interest to the readers in the distribution area of the newspaper.

By blanketing the nation with newspaper and magazine advertisements and with radio spot announcements, concentrated primarily within the week of October 8, the physicians of this country will make plain their stand on socialization of medicine and their desire to promote the best medical care possible for all the citizenship.

Members of the State Medical Association should be aware of the advertising campaign which has been authorized by the Board of Trustees of the American Medical Association

and approved by its House of Delegates. Individual physicians may also be in a position to cooperate with the press and radio in developing supplementary material for use with the advertisements and to interpret to their patients and friends the program for which the American Medical Association stands.

JAYCEES OPPOSE COMPULSORY INSURANCE

The drive for grass-roots resolutions opposing compulsory health insurance now has powerful potential assistance in the Texas Junior Chamber of Commerce.

C. J. M. Roesch, chairman of the Texas Junior Chamber's committee on public health, has announced that the Jaycee organization is preparing to launch its own campaign to put additional clubs and organizations on record opposing a trend toward socialized medicine.

In the last six months, almost six hundred Texas clubs and organizations of all types have gone on record opposing compulsory health insurance. Resolutions have been adopted by local affiliates of the American Legion, Veterans of Foreign Wars, Federation of Women's Clubs, Farm Bureau Federation, Texas Business and Professional Women's Clubs, Daughters of the American Revolution, and many others.

The Junior Chamber's campaign will extend through September, October, and November, according to Mr. Roesch, and will be pushed by the 134 member chambers of the state. Securing the resolutions has been made a major program for this year's committee on public health, he pointed out. Executive secretary of the Texas Pharmaceutical Association, Mr. Roesch said that his own association has granted him time and facilities to cover the entire state in meetings with local Jaycee groups.

"We expect to work through our local public health committees and to cooperate closely with medical and auxiliary groups already pushing this campaign so successfully," he said. "Our goal in each city will be to give as many clubs and organizations as possible the opportunity to go on record opposing this form of socialism."

Mr. Roesch expects to take about three months in contacting Junior Chambers of Commerce throughout Texas. Meantime, letters will go out shortly from State Medical Association headquarters to officers and key committeemen in the county medical societies, alerting them to the Jaycee plan and urging all possible local cooperation with Chamber representatives.

COMMUNITY CHESTS STILL SERVE

Contributors to Community Chest drives sometimes overlook the tremendous spadework which is done prior to the drives—spadework which helps to assure that each dollar given is spent to greatest advantage; spadework which helps the individual contributor to invest in a balanced, diversified plan of community service with comparatively little trouble on his part. The value of a Community Chest rests in the fact that it combines many appeals into one, that it guarantees effective use of the contributor's funds through annual budget reviews of each agency, that it assures overall planning of community services, and that it saves on campaign expense.

Donations to the Community Chest should be given with thought for the planning and effort which have gone into the setting of campaign goals. Donations should be generous to assure that the health, welfare, and character building agencies represented in the Chest budgets can operate throughout the year. Furthermore, it should be remembered that donation of time to the solicitation of funds may be even more welcome than a signature on a check.

Physicians and their wives who participate in their Community Chest campaigns as contributors and as solicitors will be performing a service to themselves as well as to their neighbors.

CURRENT EDITORIAL COMMENT

INFANTILE ECZEMA

Eczema in infants and small children is a condition which is vexing to the parents and to the physician, as well as to the patient, for despite the best efforts of a succession of physicians, the eczema may be difficult to control and probably will not be terminated until it does so spontaneously when the child is about 3 years of age. Even then, it is probable that other symptoms of the "eczema - asthma - hay fever" complex may persist—50 per cent of all children who suffer from infantile eczema develop asthma. Nevertheless, there are steps which the physician can recommend to alleviate the itching and discomfort of infantile eczema and to allay the anxiety of the parents.

Although it is usual to suspect food as the allergic irritant, the child should not be deprived of various foods just because he scratches or has a skin eruption. He should continue to have a diet sufficiently nutritious to maintain good health and promote growth; rickets and scurvy can develop when supplementary foods are removed without a substitute being made.

Stuffed toys should be avoided and unpainted wood or plastic toys used instead. Animals should be excluded entirely. Woolen and silk materials should be avoided.

Some ointments used in the treatment of eruptions will only aggravate the condition, and the parents should be warned against trying every neighborhood "cure-all." Roentgen-ray treatments should not be given in cases of infantile eczema, for permanent skin damage and even skin cancer have been observed in adults who received roentgen-ray therapy for infantile eczema. Poison ivy and other injections are worthless. Children with eczema should not be vaccinated against smallpox during the acute stage; serious or even fatal consequences may result. However, the results of diphtheria, whooping cough, and tetanus antitoxins need not be feared.

It should be remembered that some children with eczema are highly allergic to bites of insects such as mosquitoes, ants, and bees. Treatment to reduce sensitivity to such bites may be helpful. If an insect sting produces a severe reaction, the physician may find it necessary to administer adrenaline, or at least one of the antihistamines.

An effort should be made to treat the child psychologically, erasing if possible any insecurity factors which may exist. In attempting to create a wholesome environment for the child, the physician can help by reassuring the parents that the child's condition is not contagious, that there will be no scars, and that otherwise he is healthy and normal. The parents should also be warned to expect the eczema to show a cyclic pattern, with the eruptions usually being worse in the winter and better in the summer.

If a pediatrician and a dermatologist working together can gain the confidence of the parents, a number of simple treatments can be suggested for the comfort of the small patient with eczema and in time the condition may be expected to correct itself.

RALPH BOWEN, M. D., and
MANUEL G. BLOOM, M. D.,
Baylor University College of Medicine,
Houston, Texas.

This department of the JOURNAL *presents editorial comments on current items pertaining to the science, art, and practice of medicine, contributed by members of the State Medical Association and scientists closely associated with the medical profession of Texas. Invitation is hereby extended to any member of the State Medical Association of Texas to submit such discussions for this department. The discussions should not be more than 500 words in length.*

AMBULANCE PLANES LISTED

A directory of ambulance planes based in the United States, Alaska, and Hawaii has been prepared by the United States Civil Aeronautics Administration and distributed widely. Sufficient copies of the directory were made available to each state medical association so that a copy could be forwarded to each county medical society. The secretary of every county medical society in Texas is being mailed a copy of the directory, which is to be filed with the other records of the organization for reference by any member of the society.

Thirty-six ambulance planes are listed at Texas airports. A summary of the services available as well as the name, address, and telephone number of the agency providing the service is given for each plane.

ORIGINAL ARTICLES

OBSTRUCTIONS OF THE GASTROINTESTINAL TRACT IN THE NEWBORN INFANT

ORVAR SWENSON, M. D., Boston, Massachusetts

THERE are a number of congenital anomalies in the newborn infant which cause obstruction of the gastrointestinal tract. Many of these anomalies are amenable to correction and the mortality rate in these cases is related to promptness of diagnosis and proper surgical treatment.

ESOPHAGEAL ATRESIA

One of the most common of these congenital anomalies is esophageal atresia, with or without tracheoesophageal fistula. The hopeless outlook of ten years ago has changed considerably for the better. The first successfully treated cases are now eleven years old.[4]

The diagnosis of atresia of the esophagus is suggested by excessive mucus which requires frequent pharyngeal suctioning. Regurgitation occurs promptly when oral feedings are given and a bout of cyanosis usually follows. Abdominal distention of varying degree is present. The diagnosis is confirmed by inserting a small soft rubber catheter into the esophagus and demonstrating an obstruction in the proximal one-third of the esophagus.[11]

The detailed anatomic arrangement of the malformation can be gained from a fluoroscopic examination made while 1 or 2 cc. of Lipiodol are injected through the rubber catheter. The outline of a blind proximal esophagus and air filled intestine proves an atresia with a tracheoesophageal fistula of the type which comprises 94 per cent of the cases. These roentgen studies may reveal the rare forms of the anomaly, such as atresia with no fistula, a fistula to the proximal segment of the esophagus, a double fistula, or an intact esophagus with a fistula.

Operative repair of this malformation is undertaken with cyclopropane anesthesia. A right extrapleural approach is used, the fistula is closed, and end-to-end anastomosis of the esophagus is performed so that the anatomy of the mediastinum is returned to normal.[9] The older multiple stage operation—consisting of ligation of the fistula, marsupialization of the esophagus, and gastrostomy—should not be performed. It has a higher mortality rate than the primary operation and presents the almost insurmountable problem of making an esophagus for the patient. In a group of 77 patients it has been possible to do a primary anastomosis in all except 2 patients, with a total of 17 postoperative deaths.

Read before the Section on Pediatrics, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

INTESTINAL ATRESIA OR STENOSIS

Intestinal obstruction in the newborn infant may be produced by atresia or stenosis of the intestines. Vomiting of bile-stained material begins soon after birth. The amount and location of abdominal distention is determined by the site of the obstruction. Obstruction of the duodenum results in fullness in the upper abdomen, whereas atresia of the lower ileum produces marked generalized distention.

Plain roentgenograms of the abdomen without the use of contrast media are sufficient for diagnosis. The obstruction can be fairly well localized, for no gas will be present beyond the atresia. The administration of barium is dangerous because it may be vomited and aspirated. Moreover, it may become a hazard to the patient postoperatively by forming a concretion in the bowel which cannot pass through the anastomosis.

Formerly, it was considered safe to wait five or six days before operating on infants with atresia of the small intestine. However, perforation may take place as early as two days after birth, and therefore operation should be postponed only for twenty-four hours. This much delay permits the institution of Wangensteen suction to combat abdominal distention and the giving of fluids to correct fluid imbalances. The patient should be kept in an oxygen tent during this period with a high concentration of oxygen to supplement the suction in decreasing abdominal distention.

To permit careful examination of the entire intestinal tract so that multiple areas of atresia will not be overlooked, adequate surgical exposure is essential. In obstructions of the duodenum and proximal jejunum a side-to-side anastomosis should be performed. In duodenal atresia, it has been demonstrated that it is more satisfactory to perform duodenojejunostomy than gastro-enterostomy, as the latter results in loss of appetite and failure to gain weight.

The lower the site of obstruction in the small bowel, the higher the mortality rate. In low ileal atresia it may be wise to perform an ileostomy of the double-barrel type. It is surprising how well infants tolerate ileostomy. Often an ileostomy can be closed in from ten to fourteen days after the initial operation.

A gratifying number of reports of cases which have been successfully treated are being published.

MECONIUM ILEUS

In contrast to the improved results that can be reported in the majority of cases of surgical emergencies in newborn infants, little progress has been made in the treatment of meconium ileus. This disease, which is caused by pancreatic insufficiency, still has a high mortality rate. These infants ordinarily give evidence of advanced intestinal obstruction. Vomiting starts soon after birth; the vomitus contains bile; and the marked distention is progressive. The diagnosis can be reached in approximately 40 per cent of the cases on the basis of plain films of the abdomen. Prompt exploration should be performed. As a result of Farber's studies on the effect of pancreatin on inspissated meconium, enterostomies have been performed on these infants and an attempt has been made to clear the thick meconium from the lower intestinal tract by the use of pancreatic enzyme as an irrigating fluid. In a number of cases the intestinal tract has been cleared. Unfortunately, pulmonary infection eventually caused the death of most of these patients. Hiatt[3] has reported some successful cases. There have been 4 successful cases at the Children's Hospital, Boston.

MIDGUT VOLVULUS AND MALROTATION OF CECUM

Newborn infants with volvulus dependent on malrotation and faulty mesenteric attachment present the picture of an acute intestinal obstruction. When midgut volvulus is suspected, the diagnosis is confirmed with a roentgenogram of the abdomen which reveals a dilated duodenum and stomach and only small bubbles of gas beyond the duodenum. Operation should be performed promptly as the volvulus obstructs the vascular supply to the bowel and neglect results in infarction. Ladd[5] has emphasized the inadequacy of merely reducing the volvulus and the importance of freeing the malrotated cecum to relieve the duodenum of any obstructive bands as well as the cecal mesenteric vessels. The cecum must be freed completely and placed in the left upper quadrant so that obstruction to the duodenum will not recur.

We have operated on 115 such patients at the Children's Hospital in the last nine years with 28 postoperative deaths. Most deaths were caused by associated anomalies such as omphalocele, atresia or stenosis of the small intestine, and esophageal stricture.

IMPERFORATE ANUS

Imperforate anus is a common surgical emergency of the newborn infant. It is usually promptly recognized. However, the complexity of the situation is often not appreciated, since the major anomaly is frequently associated with some type of fistula.[6] Rectovesical or rectourethral fistulas are the most difficult to treat. Rectovaginal and rectoperineal fistulas carry the best prognosis, since the fistula presents an outlet for the intestinal tract. Marked distention rapidly develops in infants with imperforate anus.

It is extremely important to determine how low in the pelvis the rectal pouch is located in order to decide what type of operation is indicated. It is likewise important to ascertain whether the infant has passed gas and fecal material through the urethra with voiding or whether there has been a constant dribbling of small amounts of intestinal contents from the urethra. If the former situation exists, it is apparent that the fistula from the rectal pouch connects with the bladder, whereas in the latter group of cases it connects with the urethra. By using Wangensteen's method of inverting the infant and placing a metallic marker on the perineal dimple, the physician may determine how near the pouch is to the perineum by taking a lateral roentgenogram of the pelvis. This examination is not conclusive until after fourteen to eighteen hours of life; otherwise the gas might not have reached the rectal pouch.

If the rectal pouch is less than 1.5 cm. from the perineal skin and there is no evidence of rectovesical or rectourethral fistula, anoplasty is indicated. An abdominoperineal approach, such as that described by Rhoads,[8] should be used if the pouch is more than 1.5 cm. from the perineum or if a fistula to the urinary system is present. At the Children's Hospital 50 such operations have been performed with 2 postoperative deaths. The presence of an adequate rectovaginal or rectoperineal fistula permits the postponement of anoplasty for two or three weeks if the child is not in good condition. Ladd and Gross[6] have stressed the importance of performance of anoplasty in uncomplicated cases in the first few days of life.

DIAPHRAGMATIC HERNIA

From a therapeutic standpoint, diaphragmatic hernia in the newborn infant was an almost entirely neglected anomaly until comparatively recent years. It is not uncommon, and without radical treatment the mortality rate approached 90 per cent. Hedblom[2] has reported that 75 per cent of the patients with

congenital diaphragmatic hernia die before the end of the first month of life. This classification includes hiatus hernias. If these were excluded, the mortality rate would be higher than the one he reported.

It is not difficult to arrive at a diagnosis of diaphragmatic hernia. These infants present a fairly typical appearance, characterized chiefly by attacks of cyanosis, especially during or shortly after feedings. There is a tendency to vomit. As a result of the encroachment on the chest cavity by the abdominal organs, the respiratory rate is accelerated. On examination the heart is found to be pushed to the side opposite the hernia, with tympany to percussion in the region of the hernia. Auscultation often reveals a clear peristaltic sound. Because of vomiting and the danger of aspiration, barium by mouth is not desirable. Usually the diagnosis is clear in a roentgenogram of the chest without the use of contrast media. The hernias are far more common on the left side than on the right. Operation should be delayed only until the fluid imbalance has been corrected. During the period of preparation, distention of the gastrointestinal tract is reduced by the use of Wangensteen suction and the administration of a high concentration of oxygen.

Harrington[1] has reported 2 cases in which a transthoracic approach was used in repairing the diaphragmatic hernia, in which, because of intra-abdominal adhesions, it was necessary also to open the abdomen to reduce the hernia. Ladd and Gross[7] have advocated the use of an abdominal approach. As the capacity of the peritoneal cavity is reduced in proportion to the volume of viscera in the chest cavity, closure of the abdominal incision may be difficult. Usually, a careful closure of the layers of the abdominal wall is possible at the first operation.

Gratifying results have been achieved through operative repair for this anomaly. In the last ten years at Children's Hospital there have been 64 patients operated upon, with 7 postoperative deaths.

REFERENCES

1. Harrington, S. W.: Diaphragmatic Hernia of Children, Ann. Surg. *115:*705-715 (April) 1942.
2. Hedblom, C. A.: Diaphragmatic Hernia; Study of 378 Cases in Which Operation Was Performed, J.A.M.A. *85:*947-953 (Sept. 26) 1925.
3. Hiatt, R. B., and Wilson, P. E.: Celiac Syndrome; Therapy of Meconium Ileus, Report of Eight Cases with Review of Literature, Surg., Gynec. & Obst. *87:*317-327 (Sept.) 1948.
4. Ladd, W. E.: Surgical Treatment of Esophageal Atresia and Tracheo-Esophageal Fistulas, New England J. Med. *230:*625-637 (May 25) 1944.
5. Ladd, W. E.: Congenital Duodenal Obstruction, Surgery *1:*878-885 (June) 1937.
6. Ladd, W. E., and Gross, R. E.: Congenital Malformations of Anus and Rectum; Report of 162 Cases, Am. J. Surg. *23:*167-183 (Jan.) 1934.
7. Ladd, W. E., and Gross, R. E.: Congenital Diaphragmatic Hernia, New England J. Med. *223:*917-925 (Dec. 5) 1940.
8. Rhoads, J. E.; Pipes, R. L.; and Randall, J. P.: Simultaneous Abdominal and Perineal Approach in Operations for Imperforate Anus with Atresia of Rectum and Rectosigmoid, Ann. Surg. *127:*552-556 (March) 1948.
9. Swenson, O.: End-to-End Anastomosis of Esophagus for Esophageal Atresia, Surgery *22:*324-334 (Aug.) 1947.
10. Swenson, O.: Diagnosis and Treatment of Atresia of Esophagus and Tracheo-Esophageal Fistula, Pediatrics *1:*195-204 (Feb.) 1948.

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TREATMENT OF DIARRHEA WITH CAROB FLOUR

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THE role which diarrhea has always occupied and still occupies in infant morbidity and mortality presents a continuing therapeutic challenge. The problem has been variously approached from the standpoint of fluid and electrolyte replacement, of acid-base balance, plasma and blood infusions, chemotherapy and antibiotics, as well as from the dietary standpoint. One of us (S.R.K.) has long been convinced that prolonged withholding of oral alimentation in the treatment of diarrhea serves no good purpose unless there is associated vomiting, and this study was undertaken in an attempt to evaluate the effect of carob flour* in the treatment of the types of diarrhea which are seen in this area. The majority of cases were from Latin-American homes of low economic level. Most of the patients had received inadequate dietary and vitamin intake before developing the diarrhea for which they were hospitalized.

From the Departments of Pediatrics and Pathology of the Santa Rosa Hospital.
Read before the Section on Pediatrics, State Medical Association of Texas, Annual Session, Fort Worth, May 2, 1950.
**Supplied by the Nestlé Co., Inc.*

CAROB FLOUR

Carob flour is derived from the fruit of the carob tree, belonging to the genus Ceratonia siliqua. Known since ancient times, the bean has been eaten as a food by the poorer classes in some of the countries bordering the Mediterranean. Ramos of Barcelona, however, has been credited with first employing it in the treatment of diarrhea, allegedly having been prompted to use it because he observed that children of the poorer classes who ate the carob had less diarrhea than children of the more affluent classes who shunned this fruit. The European literature con-

tains numerous references to its use in the treatment of diarrhea in children.[2, 3, 4, 5, 6, 7]

Carob flour is a brownish powder, only slightly soluble in milk and water, having a not unpleasant taste faintly similar to cocoa. In the commercially available form (Arobon), it has the composition summarized in table 1.

TABLE 1.—*Composition of Carob Flour (Arobon).*

	%
Fat (ether extract)	0.5
Protein (nitrogen times 5.7)	3.5
Soluble carbohydrates (sucrose and reducing sugars)	49.0
Other carbohydrates (hemicellulose and pectin)	22.0
Starch	13.0
Crude fiber	6.0
Minerals (ash)	2.5
Moisture	3.5

From a practical standpoint, the low solubility of Arobon requires that feedings containing it be agitated frequently while they are being given so that a constant suspension may be maintained.

CASES STUDIED

Ninety-six patients with diarrhea, ranging in age from 2 weeks to 5 years, were studied. The age distribution is shown in table 2.

From an etiologic standpoint, the cases were classified into three groups: (1) Enteric infections, in which one or more of the recognized pathogens was recovered from a culture inoculated by rectal swap. There were 48 cases of these. (2) Cases presumably of virus etiology, in which stool cultures were negative for pathogenic organisms and in which no recognizable parenteral infection could be demonstrated. These included 21 cases. (3) Cases presumably due to parenteral infection, in which there was recognizable otitis media, furunculosis, pharyngitis, or other focus of infection commonly associated with diarrhea, and in which stool cultures were negative. These included 27 cases. A breakdown of cases, on the basis of the foregoing etiologic concept distributed according to age appears in table 2.

TREATMENT

All patients received a 5 per cent solution of carob flour (Arobon) in water or in boiled skimmed milk. The most seriously ill were given the Arobon in water initially and the water was replaced by boiled skimmed milk as improvement warranted. Those less seriously ill on admission received their Arobon in boiled skimmed milk from the outset. An aggressive attitude was maintained with reference to the early administration of skimmed milk, beginning with an intake of 10 calories derived from milk per pound of body weight (22 calories per kilogram of body weight). Seldom was the milk diet deferred more than twenty-four hours unless the child was vomiting or unable, because of disturbed sensorium, to take food by mouth. In addition, there was an increment derived from the caloric value of carob flour, which is 2.5 calories per gram.

The policy was maintained throughout of increasing the caloric intake by additions of skimmed milk, and by the time the child was taking 30 calories per pound (66 calories per kilogram) a half-skimmed milk formula was introduced. If it was apparent that the case was responding favorably (an arbitrary period of four days was set for this determination), Arobon was discontinued, and mashed, ripe banana was added to the diet. As rapidly as possible a transition to a suitable evaporated milk formula was made to prepare the child for dismissal. Limited bed capacity and the necessity for making space available for critically ill patients prohibited prolonged hospitalization, and the addition of cereals, vegetables, and other appropriate foods were accomplished under outpatient observation.

TABLE 2.—*Age and Etiologic Distribution of Cases of Diarrhea.*

Etiology	Age						
	<1 mo.	1-3 mo.	3-6 mo.	6-12 mo.	1-3 yr.	>3 yr.	Total
Enteric infections							48
Salmonella	1						
Shigella			1	2	2	2	
Proteus	2	7	8	8	1	1	
Pseudomonas			1	2			
Paracolon navy				1			
Multiple pathogens				6	1		
Presumably virus	4	2	6	4	4	1	21
Parenteral infections	4	3	7	10	3		27
Total	11	12	23	34	11	5	96

In addition, the conventional measures were employed to combat dehydration, loss of electrolytes, calcium deficit, and acid-base imbalance; and in selected cases plasma and/or blood infusions were given. A large percentage of cases received blood transfusions because of the prevalence of nutritional anemia in the economic group covered by this study. The cases in which parenteral infection was the only demonstrable cause of diarrhea received sulfonamides, antibiotics, and such surgical procedures as myringotomy and drainage of abscesses when indicated. Although facilities for measurements of blood potassium levels were not available, all severely ill patients received potassium lactate or bicarbonate by clysis and potassium chloride orally as advocated by Govan and Darrow.[1] Throughout the period of acute diarrhea, all patients were given vitamin B complex and vitamin C parenterally. When convalescence was established, multivitamin preparations were given orally.

RESULTS

Among the 96 cases studied in this series there were 6 deaths, and autopsy was obtained in 5. Of these, 1 showed gross purulent meningitis due to

Hemophilus influenzae, and another death was clearly attributed to bronchopneumonia, which was present on admission of the patient. Four deaths were presumably attributable to diarrhea.

To illustrate the severe nutritional deficiencies existing in many of the infants in this series, it might be cogent to cite the case of V. V., aged 13 months, with Shigella sonnei enteritis, who on admission weighed only 12 pounds (5.45 kilograms). This child died on the twenty-fifth day and autopsy revealed extensive ulcerative colitis.

To evaluate the results of the use of Arobon in the treatment of this series of 96 cases, it was decided to regard a result as (1) good if the stools become thicker and lessened in frequency within forty-eight hours, (2) fair if the stools were thicker and lessened in frequency in more than forty-eight but less than ninety-six hours, and (3) unimproved if it required more than ninety-six hours of treatment to produce thickening and lessening of frequency of the stools. Application of these criteria, taking into consideration the ages and etiology, is shown in table 3.

TABLE 3.—*Results of Carob Flour Therapy in Diarrhea.*

Etiology	Age	Results		
		Good	Fair	Unimproved
Enteric infection				
	<1 mo.	2		1
	1-3 mo.	4	2	1
	3-6 mo.	6	2	2
	6-12 mo.	17	1	2
	1-3 yr.	1		3
	>3 yr.	3	1	
Presumably virus				
	<1 mo.	1		3
	1-3 mo.	1		1
	3-6 mo.	3	3	
	6-12 mo.	4		
	1-3 yr.	4		
	>3 yr.	1		
Parenteral infection				
	<1 mo.	2	1	1
	1-3 mo.	2	1	
	3-6 mo.	6	1	
	6-12 mo.	4	1	5
	1-3 yr.	3		
	>3 yr.			
Total		64	13	19

Good results were obtained in 64 cases, fair results in 13 cases, and no improvement was observed in 19 cases. More than three-fourths of the cases could be regarded as improved. Corrected figures eliminating the patients succumbing to meningitis and pneumonia would be still better.

An evaluation of the results from Arobon in the cases of parenteral diarrhea was included for completeness only, inasmuch as no valid conclusions as to its efficacy can be drawn from this type of case because of the multiplicity of variables involved.

A more critical evaluation may be obtained by a tabulation (table 4) of results in cases of enteric and of virus etiology classified as "mild" (in which dehydration was not severe and in which the carbon dioxide combining power was 25 volumes per 100 cc. or more of plasma) and "severe" (in which dehydration was marked and the carbon dioxide combining power was less than 25 volumes per 100 cc. of plasma).

TABLE 4.—*Results of Carob Flour Therapy in Cases of Diarrhea with Enteric and Virus Etiology.*

Age	No. Cases	Classification	Results		
			Good	Fair	Unimproved
<1 mo.	1	Mild	1		
	6	Severe	2		4
1-3 mo.	4	Mild	2	2	
	5	Severe	3		
3-6 mo.	8	Mild	5	3	
	8	Severe	5	1	2
6-12 mo.	10	Mild	8	1	1
	14	Severe	13		1
1-3 yr.	1	Mild	1		
	7	Severe	4		3
>3 yr.	3	Mild	2	1	
	2	Severe	1	1	

In both categories and in all age groups represented in this study results were regarded as "good" in 47 cases, "fair" in 9 cases, and "unimproved" in 11 cases. The average number of hospital days from the time Arobon was started until the child was ready for dismissal was 9.9 days.

Of the 48 cases of enteric infection, 7 yielded more than one enteric pathogen on stool culture. It was deemed of interest to survey the results in these cases (table 5).

TABLE 5.—*Results in 7 Cases of Diarrhea Yielding More Than One Enteric Pathogen*

Age of Child	Organisms	Results
6 mo.	Salmonella, Proteus	Good
6 mo.	Shigella Proteus	Good
6 mo.	Pseudomonas, Proteus	Unimproved
6 mo.	Pseudomonas, Proteus	Good
7 mo.	Shigella, Pseudomonas, Salmonella	Good
7 mo.	Pseudomonas, Proteus	Good
2 yr.	Salmonella, Pseudomonas	Fair

CONCLUSIONS

In this study the use of carob flour in the early enteral treatment of 96 patients with diarrhea requiring hospitalization is reported. The majority of these patients were less than 1 year of age, though the ages ranged from 2 weeks to 5 years. In the 69 cases due to enteric pathogens and/or virus infection 47 showed "good" results in that there was an appreciable decrease in the number of and a thickening of stools within forty-eight hours. In 9 cases the results were "fair" in that a decrease in the number of and a thickening of stools was observed in ninety-six hours. Although the validity of results obtained in parenteral diarrhea is subject to question, it is interesting to note that the percentages of "good" and "fair" responses are almost identical with those observed in the enteric and virus cases. The remaining

cases were regarded as unimproved, inasmuch as stools neither lessened in frequency nor became thicker in ninety-six hours. The average hospital stay from the time carob flour (Arobon) was begun until the child was dismissed on a sustaining diet was 9.9 days.

It is our impression that carob flour (Arobon) is a useful adjunct in the treatment of diarrhea in young children.*

*AUTHOR'S NOTE: *Subsequent to the writing of this paper, Smith and Fischer, writing in the October, 1949, issue of the Journal of Pediatrics, reported their experience with the use of carob flour in 30 cases of diarrhea in infants and children. Their conclusions were as follows: "Initial and limited experience with carob flour ("Arobon") in the treatment of acute diarrheal disturbances in infants has been encouraging. Disappearance of diarrheal stools appears to be hastened, thus permitting early re-alimentation and hydration by the oral route. Further clinical trial and comparison with other hygroscopic agents is indicated."*

REFERENCES

1. Govan, C. D., and Darrow, W. C.: Use of Potassium Chloride in Treatment of Dehydration of Diarrhea in Infants, J. Pediat. *28:*541-549 (May) 1946.
2. Martin du Pan, R.: La Caroube, Ses Proprietes Antidyspeptiques, Journal Suisse de Medecin *35:*763, 1945.
3. Martin du Pan, R.: Les Proprietes Antidyspeptiques de la Ceratonia Siliqua, Bulletin de Galenica *11:* 1945.
4. Neyroud, M.: Une Nouvelle Medication Antidiarrhéique, la Farine de Caroube, Ann. Paediat. *166:*113-138, 1946.
5. Nicod, M.: De l'emploi de la Farine de Caroube (Arobon) dans le traitement des Troubles Gastro-intestineaux de l'enfant, Praxis *36:*105-109 (Feb. 13) 1947.
6. Rivier, C.: Farine de Caroube et Entérite du Nourrisson, Praxis *35:*652-653 (Oct. 3) 1946.
7. Tixier, L.: Toxicose Grave du Nourrison Guérie par la Farine de Caroube, Rev. Méd. Franc. *7:*116-120 (July) 1946.

ABSTRACT OF DISCUSSION

DR. BRUCE A. KNICKERBOCKER, Dallas: The importance of diarrhea in infant morbidity and mortality makes any new therapeutic weapon welcome.

Carob flour or Arobon according to its composition contains more than 50 per cent carbohydrate and only 4 per cent protein. Physicians have conventionally increased the protein and decreased the carbohydrate in the diet of patients suffering from diarrhea by giving skimmed, boiled milk, various commercial protein milks, and so forth, but with carob flour we are giving more carbohydrates. Why? Apparently no one really knows, but it is believed the carob bean owes its unusual mechanical and disintoxicating properties to lignin, a substance of a higher molecular weight than cellulose or pectin. The properties of lignin compare with those of pectin, and pectin, of course, is the active therapeutic principle in the use of apple, banana, Kaopectate and Kaomagma, and the various commercial products used in the treatment of diarrheas. A European investigator believes that Arobon creates conditions in the small intestines which favor the growth of the essential microbes and are detrimental to that of pathogens. He also believes that this carob meal absorbs the toxins elaborated by the pathogens.

Dr. Kaliski and Dr. Mitchell pointed out that this preparation has a low solubility which requires the frequent agitation of the mixture while it is being fed to the patient. I wonder if that in itself would not be a serious deterent to this product ever becoming widely used.

The essayists had a statistically useful series—96 cases, the vast majority of patients being less than a year old. The age factor is of course important in diarrheas. The class of patients they were dealing with subjects any therapy to the acid test.

It was interesting to note that the results of treatment were about the same regardless of the etiology. I was impressed not only by what appeared to be good results from the use of carob flour, but also by the good treatment these patients received in addition to the carob flour. I cannot help but conclude that because of the excellent handling of these patients with the conventional methods enumerated, the final results would have been good whether carob flour or some other hygroscopic agent had been used.

The low mortality is impressive; the number benefited is excellent in a series of this kind; and the average stay in the hospital of less than ten days is a better record than would be found in most hospitals.

This, I believe, is only the second study made in America on the use of carob flour in diarrheas. The authors are to be congratulated for their pioneering with this product, the well handled series, and the excellent presentation.

THE COMMUNITY AND ALCOHOLISM

The community that faces the problems of alcoholism squarely is making a sound investment in the health and happiness of all its citizens, writes Dr. Leslie A. Osborn, head of the Department of Psychiatry of the University of Buffalo School of Medicine, Buffalo.

In an article in the May 13 issue of *The Journal of the American Medical Association* Dr. Osborn cites the establishment of centers for treatment and study of alcoholism, such as that in Rochester, N. Y., and the University of Buffalo Information and Rehabilitation Center, as a move in the direction of community responsibility.

Dr. Osborn points out the frequent need of the patient in a poor physical condition for organic examination and temporary hospital care.

"The psychiatric study calls for the psychiatrist, particularly one with experience in problems at the community level. The psychiatrist is not nearly as effective alone as he is as part of a full psychiatric team—psychiatrist, internist, social worker, psychologist, receptionist, and, for in-patient work, the psychiatric nurse.

"Added to these are other workers in various cases—clergymen, judges, social agencies and the like—whose combined efforts can bring about results that no one person can achieve."

AMERICAN GOITER ASSOCIATION AWARD

The American Goiter Association again is offering the Van Meter prize award of $300 and two honorable mentions for the best essays on original work on problems related to the thyroid gland. The essays may cover either clinical or research investigations. They should not exceed 3,000 words, should be in English, and should be submitted in duplicate typewritten and double spaced to the corresponding secretary, Dr. George C. Shivers, 100 East St. Vrain Street, Colorado Springs, Colo., not later than March 1, 1951.

The award will be made at the association's annual meeting May 24 to 26, 1951, at Columbus, Ohio, providing essays of sufficient merit are presented in the competition. A place will be reserved on the program of the annual meeting for presentation of the prize award essay by the author, if it is possible for him to attend, and the essay will be published in the annual proceedings of the association.

DIPHTHERIA PREVENTION

Cases and Carriers in a Local Community

M. L. FULLER, M.D.,* Laredo, Texas

IT may appear to some a waste of time to present a subject such as diphtheria, which is not now considered a major public health problem. However, when all phases of the disease are considered, it seems to deserve more than passing mention. In this paper it will be shown (1) that diphtheria immunization is not always, if ever, permanent; (2) that diphtheria is not a disease of infancy and childhood alone; (3) that while the Schick test is the best index of immunity for mass testing, it is not 100 per cent effective; (4) that antitoxin and penicillin together given early appear to be effective; and (5) that the best measures of control are immunizations of infants (in the first year of life and a booster every three years), isolation, and prompt reporting of cases in order that contacts may be studied.

IMMUNIZATION STUDIES

In an extensive study by Bell[1] on diphtheria immunization of small children, a comparison of aluminum precipitate diphtheria toxoid and a mixture of aluminum precipitate pertussis vaccine and diphtheria toxoid was made. The aluminum precipitate diphtheria toxoid was the same product as the mixture, except that the mixture contained 10,000,000,000 killed Hemophilus pertussis organisms per dose. The children were given two doses one month apart, and a Schick test was made one year later as an index to their diphtheria immunity.

Bell reached the following conclusions:

1. Children who received two doses of unmixed aluminum precipitate diphtheria toxoid had three times as many failures of immunity against diphtheria as children who received two doses of the mixed product. Children who received only one dose of the same product had similar differences.

2. Children who received only one dose had three times as many failures of immunity against diphtheria as children who received two doses regardless of the product used.

3. Children who received both doses at an age of from 2 to 5 months had twice as many failures of immunity against diphtheria as those who received at least one of their doses at from 6 to 23 months. This was true regardless of which product was used.

4. Children who received the mixed product before they were 6 months of age had fewer failures of immunity against diphtheria than those receiving the unmixed aluminum precipitate diphtheria toxoid at any age from 2 to 23 months.

5. Children who received either product at an age of from 2 to 5 months were not protected as well against diphtheria as those who received a similar product at the age of from 6 to 23 months.

Eichelberger[2] reported cases of diphtheria from four states, Pennsylvania, New York (except New York City), Massachusetts, and California, in 1945 and 1946. The ages of patients reported range from less than 1 year to more than 55. Total cases in the four states for both years are summarized in table 1.

TABLE 1.—*Cases of Diphtheria in Pennsylvania, New York (Exclusive of New York City), Massachusetts, and California, 1945-1946.*

Age (Yr.)	No. of Cases
Under 1 year	72
1- 4	1,044
5- 9	1,330
10-14	632
15-19	379
20-24	284
25-29	236
30-34	179
35-44	213
45-54	151
55+	117
Age unknown (California only)	48

From this table it is obvious that diphtheria is not a disease of infancy and childhood alone and that no age group is immune.

It was also noted in the same study that 20 per cent of the students in junior high schools, aged from 12 to 14, and 18 per cent of senior high school students, aged from 15 to 18, had positive Schick tests. Although it was not in the program of the student body study, 43 teachers were tested; 21 were negative and 22 were positive.

Observations on diphtheria in the immunized were reported by Gibbard, Bynoe, and Gibbons.[3] They inferred that immunization against diphtheria should be considered from two angles: (1) the individual degree of immunity, and (2) immunity of the population as a whole. Generally speaking, diphtheria toxoid is an effective agent but not a perfect one, and in an epidemic a percentage of inoculated persons may be expected to contract the disease because they have not developed enough immunity to protect them from a highly virulent organism. As there are mild, moderate, and highly virulent types of disease organisms, so is there immunity on a sliding scale—from perfect immunity against the most severe infection to no immunity in the highly susceptible person.

*Director of Laredo-Webb County Health Unit.

Read before the Section on Public Health, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

Of 94 cases intensively studied by Gibbard and his associates, 23 occurred in fully inoculated but not fully protected persons; 5 deaths occurred in the series but none in the inoculated group. There were 14 severe cases, but none among the 23 inoculated were severely ill. Diphtheria occurring in inoculated persons is not a condemnation of toxoid immunization but should be an incentive to establishing a higher degree of immunity in the general population.

Some believe that an epidemic of diphtheria cannot occur in a community if 50 per cent of the population is immunized, but that does not mean that a considerable number of cases and some severe ones may not appear.

An outbreak of diphtheria in a private day school for girls with good hygienic conditions and no overcrowding was described by Fanning.[4] Ninety-four per cent of the children had been immunized and 80 per cent were Schick negative reactors. Among 322 children 18 cases occurred, 3 among 23 children who had never been immunized and 15 among 299 who had been immunized. Typing of 12 patients and 7 carriers showed the organisms to be of severe types. In this study recently immunized children seemed to have some advantage over the others; however, the morbidity rate was about the same in children immunized within five years as in those immunized earlier.

From Fanning's report it may be seen that (1) it is possible a severe outbreak of diphtheria may occur in a highly immunized community, (2) immunization fails to protect against a severe attack of diphtheria in some cases, (3) occurrence is high among Schick negative reactors. Fanning recommended that the normal immunization in infancy be followed by a booster dose every three years regardless of Schick test reading.

As reported by Schulze,[5] a program of Schick testing and diphtheria immunization was conducted among United States troops in Europe in 1946 and 1947 because of the high incidence of diphtheria (6 per 1,000 per year). It was observed in the age group from 18 to 19, that 26.7 per cent were Schick positive reactors and that the percentage of positive reactions increased in the older age group, being 35.7 per cent in the age group from 30 to 34. It was also noticed that the patients with Schick negative reaction showed more reaction to toxoid than did those with positive reactions.

Plain toxoid was used in this program giving 0.1 ml. as a test dose, followed by .5 ml. two days later, 1.0 ml. three weeks later, and 1.0 ml. three weeks later. If the test dose caused a severe reaction, no more doses were given. If no reaction resulted from the test dose but the .5 ml. dose gave a severe reaction, patients were excused from further dosage. Schulze expressed the opinion that "Diphtheria immunization of Schick positive adults using fluid toxoid in this manner is a practical and safe procedure."

Schwarz[6] reported that 16 per cent of a group of children tested from three to five years after diphtheria immunization were Schick positive reactors. Another group receiving toxoid initially and retested from eight to ten years later were 26 per cent Schick positive. Another group which received initially two doses of toxoid and which was retested from four to ten years later revealed that 22 per cent reacted positively. In 308 children retested in from three to ten years after immunization 20.8 per cent had lost their immunity.

A case of diphtheria was reported by Smith[7] in a 19 day old baby thought to be the youngest patient with diphtheria on record. The diagnosis was proved by laboratory methods. The baby had been breast fed and the mother was a Schick negative reactor. The throat culture for diphtheria was positive when the baby was 2½ months old but was negative at 4½ and 8 months of age, indicating that the baby had acquired immunity by the disease. A 5 year old brother was noted to have positive diphtheria germs, and at the time the infant was born, his brother was ill with fever, nasal obstruction, cough, and difficulty in swallowing food.

HEALTH DEPARTMENT IMMUNIZATION POLICY

For several years it has been the policy of the Laredo Health Department to immunize infants and preschool children against diphtheria by giving aluminum precipitate diphtheria toxoid combined with H. pertussis vaccine supplied by the State Department of Health. Three doses of .5 cc. each are given at four week intervals. School children up to 11 years of age receive aluminum precipitate diphtheria toxoid in two doses at four week intervals. Instead of giving .5 cc. each time, .4 cc. is given as the first dose and .6 cc. as the second. If the smaller dose causes no reaction and the arm does not get sore, children do not dread the second one so much, and if the .4 cc. does not cause a reaction, the .6 cc. dose is not likely to do so.

Older children are given three doses of plain fluid toxoid at four week intervals, a first dose of .8 cc., second dose of 1 cc., and third dose of 1.2 cc. being given for the same reason as mentioned for the aluminum precipitate product. Booster doses equal to the first dose are recommended every three years or the time closest to the three year period for the younger and older age group of school children. If more than two and a half years has elapsed since a series or booster immunizations were given in a school, assuming it would be another year before the health depart-

ment personnel returns, the booster dose is given. If a child more than 11 years of age had aluminum precipitate diphtheria toxoid the last time, plain toxoid is given as the booster dose.

Of 1,367 full series of diphtheria immunization and 364 booster doses given to school children in the school year 1949-1950, only one cold abscess and few reactions inconveniencing the child sufficiently to keep him out of school have been reported. If moderate or severe reaction occurs, the next dose is not increased but perhaps is diminished. Infants and preschool children immunized with combined diphtheria and pertussis from September, 1949, to February, 1950, numbered 73, with no reactions occurring.

CASES AND CARRIERS

Of 15 cases of diphtheria reported in the school year 1948-1949 there were 78 contacts, 33 of whom showed diphtheria-like organisms on the first culture. Fifteen of the 33 proved to be carriers as revealed by subsequent cultures showing virulent diphtheria organisms. The contacts of 4 of the 15 cases were negative on diphtheria culture; of the 15 patients only 1 was known to have been immunized three years previously. Of the 15 patients reported 2 were adults and 1 was a school age child but not in school. The other 12 patients were preschool children. The school age child and her brother, who also was not in school, showed virulent diphtheria organisms for several weeks. The brother, however, did not come down with diphtheria and it is likely that he was responsible for the diphtheria in his sister.

The information in regard to treatment was supplied by the attending physicians. One carrier showing a positive virulent test for diphtheria for several weeks was rendered negative with penicillin. Two carriers were given diphtheria antitoxin, which rendered them negative for a few days, after which they became positive again. Some of the carriers were cleared using oral penicillin. One patient was given antitoxin and penicillin early in the disease and was not positive on two cultures for release. Cultures were not taken on patients at the beginning of illness unless requested by the attending physician, but two cultures were taken for release. One 5 year old child was given 80,000 units of antitoxin and, so far as I was able to ascertain, had no other medication. This patient recovered nicely and was not positive for two cultures on release.

In this community, as perhaps in others, it is difficult to isolate the patient from other members of the family because of large families and crowded conditions. Carriers in some instances are more difficult to render negative than patients.

SUMMARY

The cases reported in this study are too few to be of much value, but in my opinion carriers are a public health hazard.

I believe, as many others do, that all children should be immunized against diphtheria in the first years of life and should have booster doses every three years until adult life. If this program were carried out, it would not be many years until a case of diphtheria would be as rare as a case of smallpox.

REFERENCES

1. Bell, J. A.: Diphtheria Immunization; Use of Alum-Precipitated Mixture of Pertussis Vaccine and Diphtheria Toxoid, J.A.M.A. *137:* 1009-1016 (July 17) 1948.

2. Eichelberger, E.: How Long Does Immunity to Diphtheria Last, Am. J. Pub. Health *38:*1234-1238 (Sept.) 1948.

3. Gibbard, J.; Bynoe, E. T.; and Gibbons, R. J.: Some Observations on Diphtheria in Immunized, Canad. J. Pub. Health *36:*188-191 (May) 1945.

4. Fanning, J.: Outbreak of Diphtheria in Highly Immunized Community, in Abt, I. A., and Abt, J. F. (editors): 1947 Year Book of Pediatrics, Chicago, Year Book Publishers, 1948, p. 119.

5. Schulze, H. A.: Schick Testing and Diphtheria Immunization of United States Troops in Europe, Am. J. Pub. Health *38:*1527-1531 (Nov.) 1948.

6. Schwarz, E. G.: Duration of Immunity to Diphtheria Following Immunization, Texas State J. Med. *37:*292-294 (Aug.) 1941.

7. Smith, C. W. R.: Diphtheria in 19 Day Old Baby—Case Report, Virginia M. Monthly *72:*128-129 (March) 1945.

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ABSTRACT OF DISCUSSION

DR. F. E. SADLER, Midland: Dr. Fuller's survey shows a high percentage of Schick positives in different age groups; also that a number having been immunized developed diphtheria. I am of the opinion that health officers should stress more to the laity the educational program of immunization and administration of booster shots every three years to overcome a mother's false sense of security that her child is immune after the initial immunization. I keenly believe that this educational program should be carried out to a greater extent in my health units. There, because of a low level of indigents, most of the immunization is done by local physicians and only through education can much follow-up work be done. I think more newspaper articles, radio programs, and addresses to parent groups on this subject should be used, especially stressing that a Schick negative reaction is not permanent.

Dr. Fuller advised penicillin therapy and also penicillin and antitoxin to clear carriers; also, a number of articles stress penicillin therapy to treat carriers. Since such widespread use of penicillin in respiratory and other infections now exists, is it possible that gradually there will be fewer carriers? I would be interested in a future survey on this for comparison, even though the dosage to cure carriers is high.

Another good point in Dr. Fuller's paper was that cases should be reported promptly in order that contacts may be studied. I am of the opinion that the contacts have not been given sufficient attention and followed through to prevent carriers.

Too long the general public has looked upon diphtheria as a disease of childhood, thus paying little attention to the disease in the older child and adult. Schulze's report on Schick testing with United States troops in Europe as a result of a high diphtheria case load, which revealed a high percentage of Schick positive reactors especially in the older age group, should give an incentive for more educational programs along this line.

MANAGEMENT OF CONVULSIVE DISORDERS IN CHILDREN

JOHN L. OTTO, M.D., Galveston, Texas

THE occurrence of the first convulsion in a child is an extremely terrifying experience for the parents. Most physicians doing general practice or pediatrics have been confronted with this situation. The purpose of this paper is to try to answer some of the many questions that arise at that time and to outline a program of examination and care for the future.

With the onset of the first seizure the question in the minds of the parents is whether or not the child is going to die. Children rarely if ever die from a single seizure. Little needs to be done other than to see that the child does not hurt himself by falling or biting his tongue. As soon as the child arouses, suitable anticonvulsives can be administered by mouth. If the seizures continue, from 2 to 4 grains of sodium phenobarbital can be given intravenously.

CAUSES OF CONVULSIONS

After the initial emergency is under control the physician is called upon to interpret the illness to the parents. Peterman[1] studied the background for initial seizures in 2,500 children and found that 33.4 per cent of the seizures resulted from acute infections, 26.3 per cent were so-called idiopathic epilepsy, 14.2 per cent resulted from cerebral birth injuries, 13.1 per cent were from miscellaneous causes, 7.4 per cent were identified as tetany, and in the remaining 5.6 per cent no cause was established. On the basis of these figures the physician could advise the parents that the chances are only one to four that this seizure is the beginning of epilepsy and that the true nature of the illness will come with further study. Children less than the age of 3 years are particularly prone to have seizures with fever, and certainly an isolated seizure does not necessarily indicate epilepsy in this group. After the age of 3 seizures accompanying fever indicate a latent tendency towards convulsions. Febrile convulsions do not require continuous medication. The child should be given anticonvulsants usually in the form of phenobarbital with the onset of each febrile illness.

When and if the recurrent nature of the convulsions is established, the question of heredity frequently comes up. Statistical studies as well as electroencephalographic surveys of relatives of known epileptic patients reveal that the incidence of seizures in children with close epileptic relatives is somewhat higher than those with no known epileptic relatives. In the light of present knowledge it has been my policy not to discourage the parents from having additional children nor to discourage an epileptic person to marry.

The cause of convulsive seizures is always uppermost in the parents' minds. Unfortunately, in most instances it is not possible to state specifically the cause. It has been the custom in the Epilepsy Clinic at John Sealy Hospital to explain to the patient or the parents that under the proper circumstances a seizure can be induced in anyone, stating that we actually induce seizures in many people each day by the use of drugs or electricity. The convulsive threshold for some persons may be lower than for others, the reason for which has not been discovered.

It is known that many different factors may contribute to the further lowering of this threshold either permanently or temporarily. The more common of these factors fall into the following groups:

1. Physical injuries to the brain such as occur during difficult deliveries, head injuries, changes resulting from infections of the brain, changes in the blood supply to the brain resulting from occlusion of vessels that sometimes accompany systemic illnesses, and congenital malformations of the brain.

2. Physiologic factors present more or less temporarily such as fever, sleep, fatigue, hunger, disturbed fluid balance, and glandular changes occurring at puberty or during menstruation.

3. Emotional factors present in the child's environment or produced by worry and anxiety over the seizures.

DRUG THERAPY

The approach to the problem of epilepsy is therefore twofold. Medicines are used to elevate the convulsive threshold, and at the same time every effort is made to discover and eliminate if possible the physical, physiologic, and emotional factors that tend to lower the threshold.

To choose the proper medicine it is necessary to determine the type of seizure. Grand mal, psychomotor, jacksonian, and focal seizures make up the group of major seizures and respond best to phenobarbital, Dilantin, Mesantoin, Phenurone, and bromides. A standard method[2] of administration of these drugs should be adopted in each case.

Because of the ease of administration and relative freedom from undesirable side effects, phenobarbital is usually recommended in infants. One dram of the elixir is added to the feeding and gradually increased until the seizures are controlled or excessive drowsiness appears, in which case the amount of pheno-

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barbital can be slightly reduced and Dilantin can be added gradually.

In older children treatment begins with .5 grain of Dilantin, which may be increased gradually until the seizures disappear or until one of the side effects such as ataxia, rash, fever, or gum hypertrophy appears. When these side effects appear, the dose of Dilantin can be reduced and one of the other drugs can be gradually added. It is important to begin the medication with small doses and gradually increase it to prevent the undesirable side effects that occasionally occur when large doses are given initially. It is particularly to be stressed that changes from one medicine to another should not be made abruptly or medication discontinued all at once lest status epilepticus ensue. In fact the most common cause of this disorder is the sudden withdrawal of anticonvulsive medication.

The minor seizures, that is, petit mal, akinetic, and myoclonic attacks, respond best to Tridione and to a lesser extent glutamic acid. The ketogenic diet is still used to some extent, but it has been my experience that whatever good is accomplished with this regime is overshadowed by the difficulty in getting the child to accept it, with the resulting bad psychologic effect on the child. It is necessary to give large doses of glutamic acid, somewhere between 12 and 24 Gm. a day, which makes this preparation difficult to administer, and since it is effective in only small percentages of cases its use is limited. Tridione remains the drug of choice in the petit mal group. It is prepared in 5 grain capsules and the dose varies from 5 to 20 grains a day. Unfortunately several fatalities from aplastic anemia have been reported with its use; therefore, it is wise to check the blood count at monthly intervals and to watch for the appearance of sore throat. Photophobia is a common harmless side effect. Some patients have both major and minor seizures; in such circumstances combinations of the above medications must be given.

The best form of psychotherapy that can be administered is to stop the seizures or to reduce materially the number or duration of attacks. The most frequent causes of failure in treatment is inadequate doses of medication and irregularity in taking the medicine prescribed. Since most anticonvulsive medications must be taken over an indefinite period it is important that the administration of the medicine be made as simple as possible. I try to control the seizures with one medicine but do not hesitate to combine two or more in an effort to attain more adequate control. Tridione must be given in divided doses throughout the day, but the medications used in the treatment of the major seizures are best given at bedtime. Usually these drugs can be given in a single dose although in a few instances better control is maintained by giving half the total dose at bedtime and half on arising in the morning.

Both children and adults find it difficult to carry their medicine with them and take it during the day at school or at work. I have obtained most consistent results by having the patient leave his medicine in a certain place at home and take it as a part of his going to bed or arising routine. Since children soon come to resent the constant nagging of their parents reminding them of their medication it is important to have them assume this responsibility as soon as they are old enough to understand the reason for taking it. After they have forgotten their medication a few times and have had a few seizures they come to realize the necessity for regularity.

OTHER FACTORS

At the same time that medical treatment is started an investigation into the physical and emotional components of the illness begins. Detailed developmental and family histories will usually reveal the presence or absence of hereditary or birth injury or other physical factors. Physical examination of the child should be detailed not only in order that the physician may inform himself as to the physical status of the child but also as a form of psychotherapy to the parents. This also holds true of laboratory examinations. Many times in order to save the parents money the physician hesitates to suggest roentgenograms or consultations; this procedure may result in the family feeling that the child has not been thoroughly examined and a decision either to seek out some charlatan or to refuse to accept the doctor's recommendations. The minimum examinations should consist of a complete blood count, urinalysis, blood Wassermann test; roentgenogram of the skull, and electroencephalogram if it is available. Depending on the information received from the history, physical examination, and laboratory tests, other special studies may be indicated.

The patient and his parents should be seen frequently in the beginning of treatment. It is important that the various studies be completed but more important that the physician have an opportunity to become acquainted with the family and thereby come to understand the setting in which the child is living. Furthermore, he should determine the attitudes of the parents toward the illness and answer the many questions that arise as the parents discuss the illness with neighbors, relatives, and teachers. By seeing the patient frequently, at least at weekly intervals at first, the physician is also able to adjust the dosage of medication and to observe any undesirable side effects.

Any physical factors such as acute or chronic infections, brain tumors, or abscesses should be correct-

ed if possible. On occasion subdural hematomas in infants can be found and removed. Rarely surgical removal of a cortical scar is indicated. Undesirable living habits such as inadequate nutrition or sleep and excessive fatigue or excitement or other unhealthy situations in the home or school should be looked into and changed.

Someone has said to give the children the medicine and the mothers the psychotherapy. The common tendency of parents is to overprotect and overrestrict the activities of an epileptic child. It is the physician's duty to combat these tendencies. The epileptic child should be allowed and encouraged to engage in all the activities of a normal child consistent with safety. It is known that children have fewer seizures when allowed to be active both physically and mentally. This is perhaps the most difficult portion of treatment for the parents to accept. Approximately 75 per cent of epileptic children on adequate treatment can live relatively normal lives, attending public schools and joining in the activities of normal children, remaining free of seizures or having so few that they do not materially influence their way of life. The remaining 25 per cent, because of concommitant physical or mental handicaps or frequent uncontrollable seizures, require special care either at home, in special schools, or in an institution. Fortunately in Texas more and more public school systems are establishing special classes which many of these children can attend and still remain at home. Also the state epileptic colony in Abilene has recently completed modern infirmary and school buildings and is assured more desirable dormitory buildings as a result of recent legislation. Even at present the educational facilities for adolescents are adequate.

If the above general suggestions regarding the care of epileptic children can be successfully carried out, the common feelings of insecurity, frustration, and being different that these handicapped children have can be largely prevented.

REFERENCES

1. Peterman, M. G.: Convulsions in Childhood, Twenty Year Study of 2,500 Cases, Am. J. Dis. Child. 72:399-410 (Oct.) 1946.
2. Price, J. C., and Otto, J. L.: Standard Treatment of Epilepsy, Dis. Nerv. System 7:345-348 (Nov.) 1946.

ABSTRACT OF DISCUSSION

DR. JAMES H. HARRIS, Marshall: A convulsion is the most alarming and serious symptom in pediatric practice. This is particularly true of the first convulsion that a child has. To physicians who see the child in the home, the most pressing matter is immediate treatment. Dr. Peterman* has outlined a program for immediate treatment which is effective: (1) do not give opiates; (2) reduce fever with hydrotherapy and antipyretics; (3) give phenobarbital by mouth or sodium phenobarbital subcutaneously; and (4) if such therapy is not effective in eight hours, give Avertin, 75 mg. per kilogram of body weight by rectum. If the patient is having a convulsion when seen by the physician, I see no objection to giving inhalation anesthesia until he relaxes.

The next step is to diagnose the disease of which the convulsion is merely a symptom. The common illnesses that cause acute nonrecurrent convulsions in my practice are pneumonia, measles, tonsillitis, high environmental temperatures, roseola, and pyelitis, all of which are extracranial; meningitis, encephalitis, cerebral abscess, and lockjaw, which are intracranial; tetany, which is a metabolic disease (and may be recurrent); acute glomerular nephritis in which there is acute cerebral edema; and intracranial hemorrhage.

If the disorder is recurrent, however, the problem is different. The commonest forms here encountered are epilepsy, either organic or idiopathic; tetany; and syphilis. I believe an electroencephalogram should be done on every patient suspected of having epilepsy. If the clinical impression is confirmed, there is then no hesitation in starting on medicines that will have to be taken indefinitely. A satisfactory electroencephalogram will establish the diagnosis of epilepsy in 85 per cent of the cases. A roentgenogram of the skull may reveal unsuspected toxoplasmosis, calcification, or other lesions. If tetany is suspected, a Sulkowitch test will give a rough clinical assessment of the calcium metabolism.

In the May, 1948, *Journal of Pediatrics,* a group of workers in Baltimore reported a study that indicates Benzedrine and Dexedrine sulfate are useful in the control of petit mal epilepsy.† They recommended its use in combination with phenobarbital or Dilantin to prevent the "breaking through" of grand mal seizures as sometimes happens when the petit mal attacks are brought under control. The same workers reported recently‡ on the use of Phenurone in the treatment of epilepsy and recommended its use in the treatment of psychomotor seizures. These constitute a small proportion of all epilepsy and are least likely to respond favorably to the drugs in common use. Although side reactions were noted and the drug was discontinued in some instances, the authors believed it to be superior to Dilantin and Mesantoin in controlling psychomotor seizures.

One word about the ketogenic diet. Dr. McQuarrie wrote that "it has been demonstrated beyond question that fasting causes cessation of grand mal seizures in a majority of epileptic children, the effect usually manifesting itself shortly after ketosis appears on the third day and lasting thereafter for the duration of the fast. A strongly ketogenic diet . . . has a similar anticonvulsive effect after ketosis has developed. . . . Establishment of a negative water balance, either by restricting the intake or increasing the output, intensifies the anticonvulsive effects of the ketogenic regimen. . . . The chief value of fasting is that it can be used to determine in the shortest possible time whether or not the patient with severe epilepsy is likely to respond favorably to the ketogenic regimen. . . . It has long been recognized that exclusive drug therapy may not only fail to prevent, but may increase the tendency to mental deterioration in the epileptic patient. It should be noted that the dietary regimen does not have this deleterious effect . . . in suitable cases may actually help to correct the underlying cellular defect to some degree."§

*Peterman, M. G.: *Convulsions in Childhood, Practical Considerations, South M. J. 41:62-66 (Jan.) 1948.*

†*Livingston, S.; Kajdi, L.; and Bridge, E. M.: Use of Benzedrine and Dexedrine Sulfate in Treatment of Epilepsy, J. Pediat. 32:490-494 (May) 1948.*

‡*Livingston, S., and Kajdi, L.: Use of Phenurone in Treatment of Epilepsy, J. Pediat. 36:159-164 (Feb.) 1950.*

§*Nelson, W. E.: Mitchell-Nelson Textbook of Pediatrics, ed. 4, Philadelphia, W. B. Saunders, 1945, p. 1096.*

SOME PRACTICAL POINTS IN PREVENTING MALADJUSTMENTS IN CHILDREN

JOHN G. YOUNG, M.D., Dallas, Texas

A PEDIATRICIAN should consider the child wholly and as an entity in relationship to his whole environment. Sometimes the failure to consider the emotional and social aspects of the child may lead to an incomplete or wrong conclusion to a pediatric problem. Prevention in many fields is stressed, and along with prevention of infections and malnutrition should go the prevention of social and emotional maladjustments. Preparation for the specialty of pediatrics should include thorough training in the growth and development of the child—physical, mental, and emotional.

The need for such training from the parents' standpoint can be fully appreciated by the pediatrician in active practice. Also, the underlying desire to become equipped can be seen among pediatric residents in training. The desire has been demonstrated every year by the resident staff at the Children's Medical Center in Dallas, and the opportunity for such training is eagerly seized when offered. To neglect this training and to have a group of pediatricians who cannot properly aid and counsel parents in the emotional problems of growth which are common to a pediatrician's office is to leave a void in the care of those who are often in deep need.

PREVENTIVE TECHNIQUES

Proper Attitudes.—Attiudes are often much more important than the techniques employed in the emotional guidance. If the attitudes are right, there is usually less haste and blustering, more calmness and helpfulness. The techniques in meeting problems or even crises that may arise will be satisfactory in most cases if the adult's attitude toward the child is a desire to aid him over the long haul and not just to do immediately what someone has said should be done.

With a good attitude on the part of the parent the child secures an unspoken feeling of comradeship and good will that no simple or complex techniques can transmit. Attitudes of reliance, helpfulness, and good will which are fostered will many times prevent problems or crises from arising. Differences in attitudes are demonstrated innumerable times each year to the pediatrician dealing with the parent-child relationship. Some parents and their children are ever in the midst or on the brink of difficulties in their relationships and others because of wholesome attitudes seldom are faced with difficult problems. This cultivation of proper attitudes should be the first element in the prevention of maladjustments.

The need to begin early the prevention of wrong attitudes and training in the right ones is important. Much has been written about the transfer of security, contentment, and a sense of well-being to even the nursing infant. Some writers stress the importance of breast feeding as a practice of mutual benefit to mother and child. They further urge that the father have a place in the new baby's daily routine so that both parents will have a share in the benefits arising from the baby's care.

Good attitudes should be established early and should be continued. It is well believed that emotional patterns are made early, and the frequent complaint of the psychiatrist, who sees those in whom the wrong emotional patterns have been developed, is that nothing preventive or remedial was done until too late or almost too late.

Instruction and training should be given to expectant parents stressing attitudes that are natural or that become second nature. The great difficulty is that parents-to-be often are not receptive, are too anxious, or become too tense over the coming responsibility to assimilate properly and profit from the teaching.

Dividends.—The parents' knowledge of what the child desires as a goal, dividend, or a satisfaction is desirable. Children work on a dividend basis and the parents should ascertain what this dividend, goal, or satisfaction is. If possible, he should let it accrue to the child for doing those things that are socially and emotionally good. The goal may be hidden from some parents, but with study and insight they may frequently find it in their children. It may not be what adults commonly think of as a dividend. To some children punishment may be a goal, for they receive a dividend in attention, be it good or bad, and they may be willing to suffer the physical discomfort to secure attention. This condition is not uncommon, but failure to recognize it may be misleading. It is fairly well established that human beings usually will not continue in any repeated action that does not in some way bring a reward.

Allowing the desired goal or dividend to accrue for doing so called "good" things and for not doing "bad" things may require more thought than some parents can muster. Failures are numerous. Many children are reared on the theory that they get what they want (dividends) if they are asocial, antisocial, or just plain "bad" enough.

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Security.—Dependability or security of environment is essential. The parent-child relationship is improved when the child has consistent and dependable parents. If the attitudes are ever changing, the child loses his sense of security. Many parents vary much from day to day or even hour to hour. Many children are able to do certain things without question at one time and are punished, scolded, or made to feel inferior for doing the same things at another time. From evening to evening many fathers may be entirely different persons to their children. The child of such a parent may be uncertain as to what the present attitude is, what can be done, whether punishment will ensue, or whether the action will be mutually agreeable. Illustrative examples could be multiplied, however, suffice it to say that a child in these or similar circumstances has no dependable environment, which he needs to become emotionally adjusted.

Love.—Love which exists and is shown in the parent-child relationship aids greatly in adjustment. Of course, parents usually love their children, but the children may not realize it. The relationship is usually good when children are aware of their parents' love; there is a mutual trust and faith that frequently bridges over many poor techniques. It sometimes requires a great deal more than just verbally expressed love to bridge the gap, for there is nothing poorer or weaker than spoken love alone. The parents by their actions should show the understanding, helpfulness, aid, and reliance so much needed by the child and thus give the foundation to their verbal expression. Many examples of this deficiency are seen yearly in the private practice of pediatrics and in clinics for child guidance. To evaluate properly and then to advise how to carry out a parent-child relationship of mutual love on a foundation of aid one for the other may prevent many small problems that might grow into major maladjustments.

As an example, a boy 11 years of age was seen because he had been under the supervision of police authorities two or three times. The psychiatrist talked to the mother and the son for a time and then to the son alone. It is surprising at times how much can be learned by conversation with a child whose confidence is gained without the parent present.

He said, "If I could get my food I wouldn't live at home."

"Why not?" he was asked.

"Because nobody loves me at my house." This was said with tears in his eyes.

Later the mother was told of his feelings and she beat him unmercifully for what he had said. Since then he has been in a reformatory twice. Perhaps the right kind of love in that family would have brought better results.

Outside Influences.—Influences outside the family may hinder good adjustment. The gregarious instinct and the pressure of the herd may play an important part, which is felt to a greater extent, perhaps, during adolescence. To recognize this influence and meet it is of importance in the parent-child relationship and also in the child's adjustment outside the home. Stiff-necked parents may be unyielding in their restrictions at times, often considering the older child as being much younger and often determinedly holding as ideals and limits for the child their own restricted actions when they were the same age. This attitude results in conflicts and at times almost open hostility between parent and child.

To prevent a bad relationship it is necessary for parents to recognize that a 14 year old child does not act and respond as does a 6 year old. Responsibilities and freedoms should go with older years; also, parents should learn what is "being done" in these times. Desirable standards can be maintained if a proper relationship exists, but it is difficult to buck the pressure of the herd without good relationship. Adolescence should mean freedom with control.

The pressure of the herd instinct may be relieved and turned toward more acceptable channels if a concerted attitude and action is taken by the parents of the youngsters who more or less consistently run together. This action may take various patterns, but if there is a meeting of minds among the parents and their decision is given to the members of the herd by individual parents, the course of the herd may be changed. If concerted action is not taken, the child may feel oppressed or restricted to the point at which authority is flaunted or a martyred attitude is taken. Instead of "Everyone can do it except me," his attitude should be, "No, we don't do that." Such situations may be handled in many ways, but it must be remembered that individual action after a concerted decision has been made is best.

Problems of adolescence can rarely be solved if neglected until they reach a difficult stage; therefore, preparation is rightly begun early. If the parent-child relationship has been wholesome and the steps outlined up to this point have been taken, the problems of adolescence are diminished. If the attitudes have been good and dependability and consistency in giving help has been practiced, the child again most frequently will rely for help on those proved sources. The problem is more than half solved with seeking aid in right places. Adolescent boys and girls should be advised, "Ask help in time of trouble from those who love you most," and that should be the parents.

SUMMARY

Only a few points in preventing maladjustment have been covered briefly. No one formula is a panacea. Parents should be taught and trained, and it is

the obligation of the pediatrician to aid them because of his enviable position as doctor of the child and adviser to the parents in the growth and development of the child. The rewards often are hard to see and often are great. The expenditure of time may occasionally seem misspent.

Freedom from many conflicts and complexes comes with good emotional adjustment, and happy children are the result. Happiness in childhood usually means happiness in adulthood.

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ABSTRACT OF DISCUSSION

DR. PAUL R. MEYER, Port Arthur: I believe strongly that insufficient attention is placed on the importance of preventing maladjustments in children.

The so-called self demand schedule may have its good points, but it encourages parents to believe that the child should be his own judge and jury. Although the method changes, the principle is adhered to as the child grows older, and he then learns that outbursts pay the best dividends. The child, especially in one child families, becomes overindulged and, upon reaching school age, comes to an obstacle which he has not learned to hurdle: a teacher or another child who cares not whether he is pleased. The child has not been taught previously to conform to the rule of the majority or how to accept disappointments.

Pediatricians are frequently consulted by anxious parents whose children complain early in the morning of various ills which disappear if they are allowed to stay home from school. Another common cause of maladjustments in the two or three child family is the failure of the parents to recognize the beginning of jealousy in the older child. Failure of the pediatrician to call attention to this possibility and thus prevent its occurrence is all too frequent. The 3 or 4 year old who suddenly starts soiling his clothes after a brother or sister has arrived is usually jealous.

As Dr. Young has pointed out, children are consistent; it is adults that are inconsistent. No child is born bad, and every problem child has at least one problem parent. The inexcusable punishment of the 2 year old child who unintentionally breaks an antique vase while being allowed to play in a grown up living room and to whom the value of the vase means nothing is as much a cause for maladjustment as is the child's living in a home with parents totally unsuited for each other and in which there is constant bickering. The forcing of a child to eat certain foods while he sees one of the parents refuse it, the telling of untruths which the child easily finds out to be untruths, the frightening of the child in order to get him to behave—all lead to a feeling of insecurity. Yet all of us, regardless of age, require security along with love and affection to be happy and adjusted to the vicissitude of life.

SURGICAL TREATMENT OF ESOPHAGEAL LESIONS

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RAPID advances have been made in the field of esophageal surgery during the last decade. It is now possible to perform satisfactory one stage procedures for a variety of esophageal lesions including congenital atresia with tracheoesophageal fistula, acquired tracheobronchoesophageal fistula, acquired perforation or rupture of the esophagus, stricture, diverticulum, cardiospasm, benign tumor, and carcinoma. The aim of the surgical procedure utilized for any of the lesions is to establish esophagogastric continuity so that the patient can swallow his food in as normal a manner as possible. Ten years ago radical surgical procedures on the esophagus were either unheard of or looked upon with fear and discouragement because of the attendant high mortality and morbidity. Today it is possible to resect short segments of the esophagus and perform an end to end anastomosis, to resect any length of the esophagus including almost the entire esophagus and to perform a high intrathoracic or cervical esophagogastrostomy, to perform an end to end anastomosis for a congenital esophageal atresia, to remove diverticula in a one stage operation, and to perform various plastic procedures for stricture.

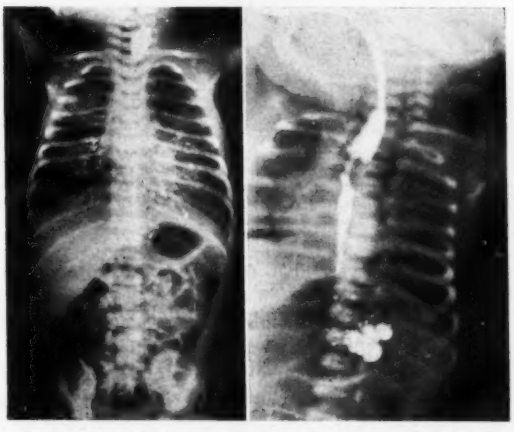

FIG. 1a. Lipiodol filling an atresia of the esophagus with overflow into the trachea, demonstrating tracheoesophageal fistula.

b. Results after division of the tracheoesophageal fistula and end to end anastomosis of the esophageal segments in the case illustrated in *a*.

Read before the Section on Surgery, State Medical Association of Texas, Annual Session, Fort Worth, May 2, 1950.

CONGENITAL ANOMALIES

The most common congenital anomaly consists of an atresia of the esophagus associated with a tracheoesophageal fistula. Either the atresia or tracheoesophageal fistula alone may be present in the absence of the other. Rarely, an esophageal web may be present occluding the lumen. There may be an agenesis of the distal esophagus.

The diagnosis of congenital esophageal atresia associated with a tracheoesophageal fistula is easily made.

FIG. 2. Roentgenogram showing an acquired bronchoesophageal fistula in a 53 year old man who gave a history of choking on liquids since the expectoration of hard rocks from a tuberculosis gland two years previously. (Courtesy of Dr. L. J. Kleinsasser, Veterans Administration Hospital, Dallas, Texas.)

The usual story is that of a newborn infant who coughs and chokes on its own saliva or on attempts at feeding. In such an infant the passage of a small catheter into the upper esophagus will prove an obstruction. This may be visualized under the fluoroscope. The physician may readily introduce Lipiodol into the proximal esophageal segment by means of a catheter (fig. 1a). Barium should never be used. The presence of air in the stomach and intestines when a blind upper segment is present proves a tracheoesophageal fistula. If air is not present in the stomach and intestines, but a blind upper esophageal segment has been demonstrated, the physician is dealing with either an atresia without the tracheoesophageal fistula or the distal segment of the esophagus is represented only by a cord. A tracheoesophageal fistula may be present in the absence of an atresia. In such an infant the catheter will pass normally into the stomach. However, installation of Lipiodol into the esophagus with the infant in the prone position will demonstrate a tracheoesophageal fistula.

Inasmuch as a congenital atresia with a tracheoesophageal fistula is incompatible with life, immediate surgery is indicated. The surgical treatment consists of (1) division of the tracheoesophageal fistula and

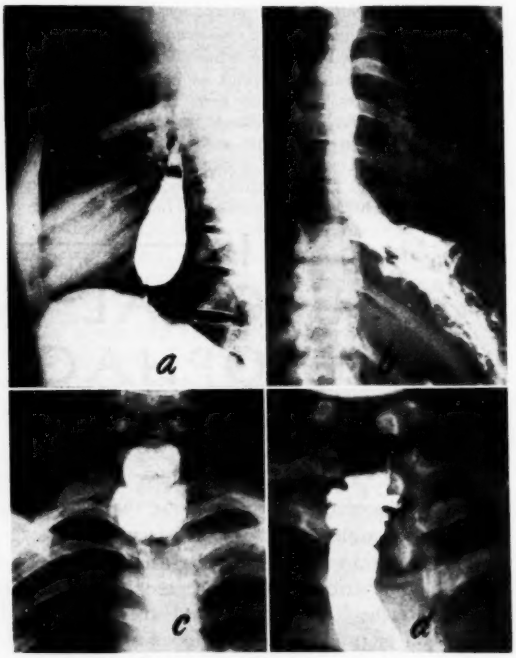

FIG. 3. Stricture of the esophagus caused by ingestion of lye.
a. Stricture of the lower esophagus with proximal dilatation.
b. The result after esophagogastrostomy in the case illustrated in *a*.
c. Complete stricture of the esophagus of thirteen years' duration.
d. The result after a plastic procedure consisting of longitudinal incision through the stricture in *c* and suture of the mucosa transversely.

(2) the establishment of normal continuity of the esophagus. This can usually be done in a one stage operation in which an end to end anastomosis is performed between the esophageal segments after division of the tracheoesophageal fistula (fig. 1b). In those instances in which either the atresia or the tracheoesophageal fistula is present alone the problem is easily handled surgically. In a few instances the gap between the esophageal segments is so long as to preclude an end to end anastomosis or the distal seg-

ment may be represented only by a cord. In such instances the tracheoesophageal fistula should be divided and the proximal esophagus brought out above the clavicle anteriorly as an esophagostomy. At a later date esophagogastric continuity can be reestablished

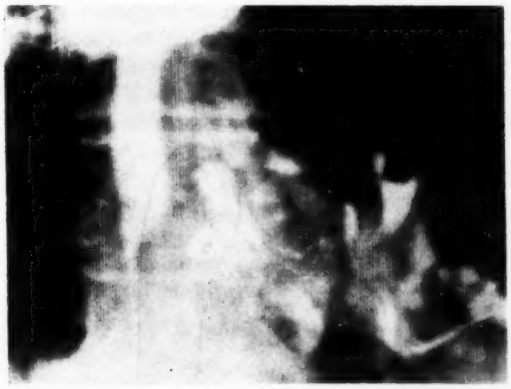

FIG. 4. Perforation of the esophagus demonstrated by Lipiodol in the esophagus and leak into the mediastinum and pleural cavity.

by using a jejunal transplant or performing a high intrathoracic or cervical esophagogastrostomy.

Gastrostomy for feeding purposes should not be done until the tracheoesophageal fistula has been divided because of the danger of regurgitation of gastric contents into the tracheobronchial tree.

ACQUIRED FISTULAS

Acquired tracheoesophageal or bronchioesophageal fistulas may be secondary to trauma such as caustic burns of the esophagus, the erosion of tuberculous

FIG. 5. Pharyngoesophageal diverticulum which can be removed in one stage.

glands into these structures, or carcinoma. Those fistulas secondary to caustic burns or the erosion of tuberculous glands may be corrected surgically (fig. 2). If the fistula has not been present long enough to produce pulmonary suppuration, simple division of the fistula is usually sufficient to effect a cure. If a stricture of the esophagus is present, a plastic procedure or resection of a portion of the esophagus may be necessary to establish normal esophagogastric continuity. If pulmonary suppuration has occurred as the result of a fistula of long standing, pulmonary resection may also be indicated. In those cases in which the fistula is secondary to the erosion of a tuberculous gland the patient usually gives a history of the ex-

FIG. 6. Leiomyoma of the esophagus with a filling deformity. This condition was treated with resection of 4 cm. of esophagus and end to end anastomosis. (Courtesy of Dr. J. A. Carswell, Veterans Administration Hospital, McKinney, Texas.)

pectoration of small hard particles which consist of pieces of a calcified lymph node. A simple division repair of the fistula usually is sufficient in these cases.

BENIGN ACQUIRED STRICTURES

Acquired strictures of the esophagus are usually secondary to caustic burns, peptic ulceration, trauma, surgical procedures, and tuberculous glands. The stricture may be partial or complete (fig. 3). Surgical treatment is indicated in those cases which do not respond well to the more conservative measures such as dilatation. Various plastic procedures, local excision with either end to end anastomosis or esophagogastrostomy, or esophagogastrostomy without excision may be carried out. In certain instances resection of the esophagus and the substitution of a piece of jejunum for it may be done.[3]

PERFORATION OR RUPTURE

Spontaneous perforation of the esophagus is not a rare condition, judging by the frequency of reports recently made in the literature. Perforation of the esophagus may be spontaneous in a presumably normal esophagus; it may be secondary to trauma following the ingestion of a foreign body or a surgical procedure such as dilatation; or it may be due to perforation of a peptic ulcer. The esophagitis associated with a diaphragmatic hernia may antedate the perforation. The patient's history is important in determining the cause of the perforation. The history of ingestion of a foreign body or operative manipulation will establish trauma as the cause. The previous history of symptoms of ulcer of the esophagus or of an esophagitis associated with an esophageal hiatal diaphragmatic hernia should make the physician suspicious of the underlying cause. In most of the cases of spontaneous perforation of the esophagus reported in the literature there has been no background in the history that would suggest disease of the esophagus.

Although the symptoms and signs differ according to the cause, any esophageal perforation produces mediastinitis and not infrequently an empyema also. Prompt surgical attention is necessary in addition to the liberal use of various antibiotics. The diagnosis can readily be made on the basis of the history and the roentgen observations. These latter will reveal air or fluid within the mediastinum behind the heart or in the neck. A swallow of radiopaque oil with fluoroscopic observation may demonstrate the esophageal perforation (fig. 4).

The treatment of esophageal perforation varies with the cause, the size of the perforation, and the duration. In any case the mediastinitis or empyema must be treated by drainage. If the perforation is seen early, whatever its cause may be, operative closure of the opening in the esophagus may save the patient a long and severe illness. Spontaneous perforation or rupture, presumably in a normal esophagus, usually occurs during vomiting, most frequently after a bout of alcohol ingestion. These perforations are usually large and the mortality almost 100 per cent if the condition is treated conservatively. The treatment of choice consists of prompt thoracotomy, wide opening of the mediastinal pleura, closure of the esophageal defect, adequate drainage of the mediastinum, and closure of the chest with intercostal tube drainage.[4] This procedure must be carried out in spite of the patient's poor condition. The large opening in the esophagus leads to a rapidly extending mediastinal infection which can only end fatally in a short period of time. Attempts to drain the local infection by mediastinotomy or pleural drainage during the acute stage have generally been unsuccessful in the case of spontaneous perforation in a presumably normal esophagus.

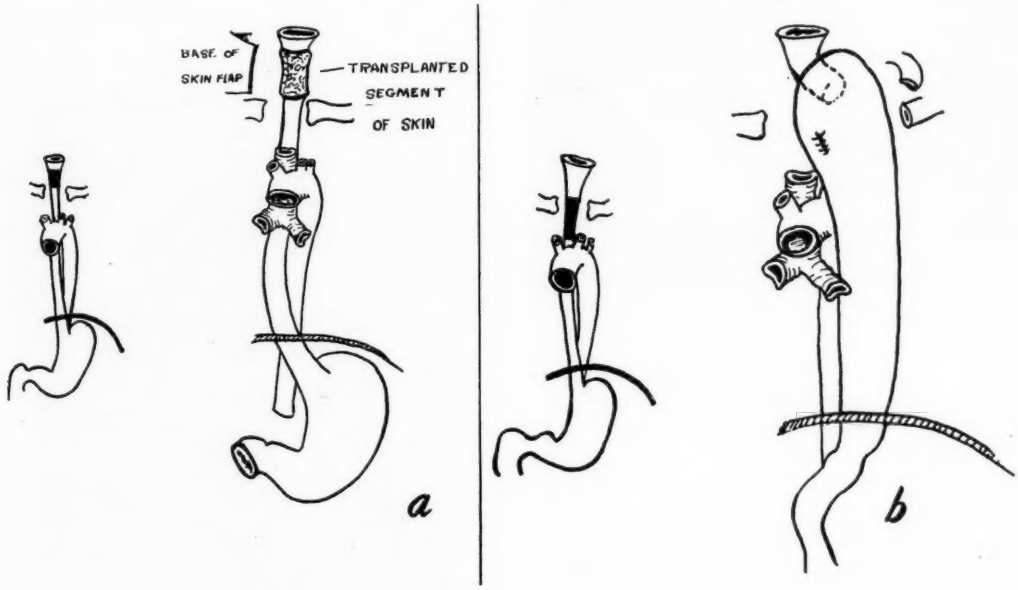

FIG. 7a. Drawing showing a method of surgical resection of a carcinoma in the cervical segment of the esophagus and reconstruction of continuity by means of a skin tube after the method of Wookey. b. Drawing showing a method of resection of a carcinoma in the upper fourth of the esophagus and a cervical esophagogastrostomy. [Both drawings after Sweet, R. H.: Treatment of Carcinoma of Esophagus and Cardiac End of Stomach by Surgical Extirpation; 203 Cases of Resection, Surgery *23*:952-975 (June) 1948.]

DIVERTICULA

A diverticulum of the esophagus may be pharyngoesophageal (fig. 5) or epiphrenic in location. The traction diverticula due to a tuberculous gland or trauma may be located anywhere along the extent of the esophagus. Diverticula vary as to size and extent, but as time goes on they usually progressively enlarge. The weight of the diverticulum and its contents pull it in a downward direction so that it narrows or compromises the normal esophageal passage. Its own opening progressively enlarges. A varying amount of inflammation occurs in the wall of the diverticulum, and ulceration and perforation may occur as complications. One stage surgical removal can now be done with a high degree of success without fear of mediastinitis.[1]

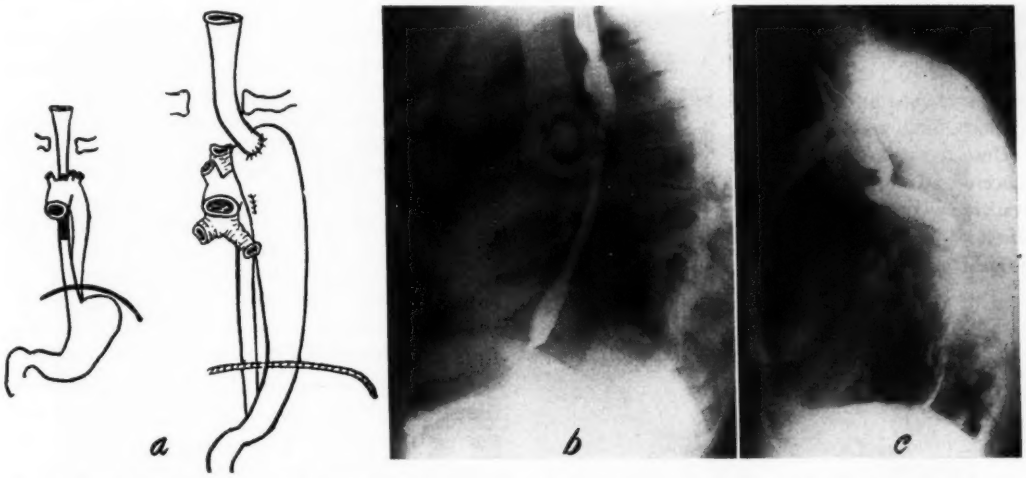

FIG. 8a. Drawing demonstrating a surgical procedure for treatment of a carcinoma in the middle half of the esophagus. [After Sweet, R. H.: Treatment of Carcinoma of Esophagus and Cardiac End of Stomach by Surgical Extirpation; 203 Cases of Resection, Surgery 23:952-975 (June) 1948.]

b. Roentgenogram of a carcinoma in the middle half of the esophagus.

c. Results in the case illustrated in *b* after resection and a high esophagogastrostomy on a level with the manubrium.

CARDIOSPASM

In those cases of severe cardiospasm or achalasia of the esophagus in which repeated dilatation of the esophagus does not produce the desired results or in those cases in which pulmonary complications or severe anemia are complications, a plastic procedure at the esophagogastric junction to enlarge the opening may be indicated and can be done transthoracically at a low risk.

BENIGN TUMORS

Benign tumors of the esophagus are relatively uncommon. They may be divided into two main groups: the mucosal and the extramucosal tumors. The mucosal tumors are pedunculated and arise from the submucosa. Usually they are fibrolipomas or fibromyxomas covered by normal epithelium. The extramucosal tumors arise from the muscle of the wall of the esophagus and usually do not involve the mucosa. These are commonly leiomyomas.

Growth of these tumors is usually slow; therefore, they may attain considerable size before giving rise to serious symptoms. Symptoms produced by these tumors are dysphagia, pain, regurgitation of food, cough, and dyspnea. A pedunculated tumor may be regurgitated itself into the mouth. The diagnosis is readily made by the roentgenographic and esophagoscopic examination of the esophagus (fig. 6).

Although the indications for surgical treatment of benign tumors are not as urgent as for malignant lesions of the esophagus, surgical removal is indicated. These tumors are usually large when first seen and therefore act as mediastinal tumors by compressing the esophagus and the adjacent mediastinal organs. It is possible that these tumors may undergo malignant change. The pedunculated tumors may be readily removed through a cervical or transthoracic esophagotomy. The intramural tumors may be shelled out from the wall of the esophagus, preserving the intact mucosa, the wall being reconstructed after removal of the tumor. In a few instances the mucosa will be damaged in the removal of the leiomyoma; this damage may necessitate a resection and an anastomosis.

Small pedunculated tumors may be removed through the esophagoscope by means of a snare and cautery.

Esophageal cysts do occur and may be readily re-

moved. They produce symptoms by pressure on the esophagus and adjacent mediastinal organs such as various parts of the tracheobronchial tree.

CARCINOMA

Cancer of the esophagus is of more frequent occurrence than are all malignant tumors of bone and is even more common than cancers of the lip, tongue, larynx, or kidney. It constitutes about 4 per cent of the deaths from cancer of the esophagogastrointestinal tract in the United States (gastric cancer 36 per cent[5]) and from 5 to 10 per cent of all carcinomas in males, being surpassed in frequency only by carcinoma of the stomach, lung, and rectum.

Until ten years ago there was justifiable pessimism concerning esophageal cancer. Surgical procedures available were mutilating and were staged procedures. Often the patient died from his cancer before the surgical plan of attack could be completed. The first

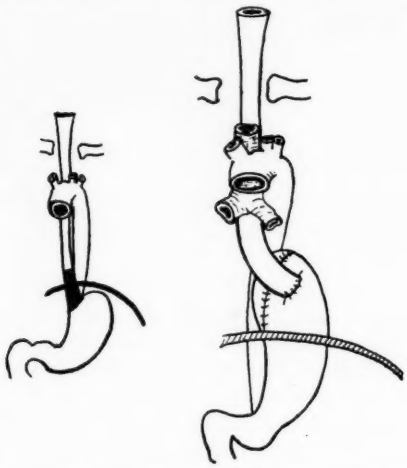

FIG. 9. Drawing demonstrating a surgical procedure for treatment of a carcinoma in the lower fourth of the esophagus. [After Sweet, R. H.: Treatment of Carcinoma of Esophagus and Cardiac End of Stomach by Surgical Extirpation; 203 Cases of Resection, Surgery *23*: 952-975 (June) 1948.]

successful resection and esophagogastric anastomosis for esophageal cancer was reported by Adams and Phemister in 1938.[2] During the ensuing ten years, this operation has been extended and modified so that now it can be applied in the treatment of carcinoma of the esophagus at all levels with the exception of the cervical segment. The morbidity and mortality of this surgical procedure have reached acceptably low levels.

Although sufficient time has not elapsed for a statistical study of end results, reports which are available[6] suggest that carcinoma of the esophagus treated by radical resection will behave like carcinoma elsewhere in the body (lung and stomach) and that there will be enough cases of three to five year survival to justify the use of the operation in the hope of effecting a cure. Because of the pitiable plight of the patients who are unable to swallow because of complete esophageal obstruction, it is justifiable to make an effort to remove the cancer in every case, however hopeless the prognosis may seem. One of the major benefits of the operation is the restoration of esophagogastric continuity and the ability of the patient to swallow food. Many such patients may live in relative comfort without any recurrence of dysphagia for as long as two or more years after resection and an esophagogastrostomy.

The obscurity of symptoms all too frequently results in a late diagnosis. Most patients coming to surgical treatment have had dysphagia for a period of six months or more. The medical profession as a whole must be alert to the possibility of esophageal cancer to make the diagnosis early. Any patient with dysphagia deserves careful roentgenographic and esophagoscopic investigation before any treatment is instituted. These two diagnostic procedures will readily reveal the presence of an esophageal carcinoma.

The surgical procedure utilized for a carcinoma of the esophagus varies according to the region in which the tumor lies. The restoration of continuity after resection of a carcinoma in the cervical segment of the esophagus requires the substitution of a turned in portion of skin for the excised portion of esophagus. This may be accomplished by the method described by Wookey[7] (fig. 7a). If the carcinoma is located in the upper fourth of the thoracic segment (in the superior mediastinum), a one stage esophageal resection with a cervical esophagogastrostomy is performed by means of two incisions. Through a left thoracotomy incision the entire esophagus from the base of the neck to the cardia is dissected free and resected. The thoracic incision is closed and a second incision is made over the lower cervical region anteriorly. The esophagus is dissected out through the incision, the fundus of the stomach being brought up into the cervical region from the chest, and the esophagogastric anastomosis is done within the neck above the level of the clavicle, actually in the subpharyngeal region (fig. 7b).

A carcinoma in the middle half of the esophagus (extending from the superior border of the aortic arch to the inferior pulmonary vein) is treated by esophageal resection and a high intrathoracic esophagogastrostomy (fig. 8). Whether the anastomosis is done superior or inferior to the aortic arch depends on the location of the tumor. Any cancer which lies at least in part behind the aortic arch requires a high level of resection to remove the cancer adequately. Esophageal

carcinoma characteristically extends submucosally so that wide resection is advisable in an effort to extirpate the lesion.

A carcinoma in the lower fourth of the thoracic segment—that is, that portion lying between the inferior pulmonary vein and the stomach—is treated by resection of the esophagus and a portion of the stomach lying along the lesser curvature and a low intrathoracic esophagogastrostomy (fig. 9 and 10). Without pathologic examination it may be difficult to determine the exact origin of the tumor, whether it be esophagus or stomach. From a practical standpoint, however, the operative technique is the same in either case.

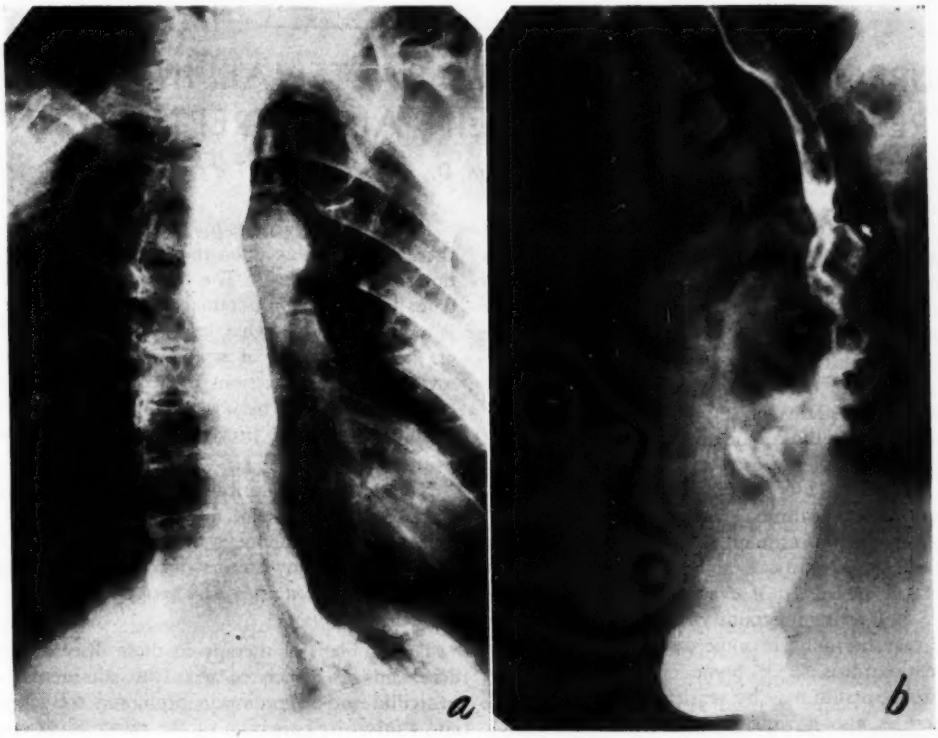

FIG. 10a. Roentgenogram of a carcinoma in the lower fourth of the esophagus.

b. Results after resection and a low esophagogastrostomy in the case illustrated in *a*.

SUMMARY

As a result of rapid advances in the field of esophageal surgery in the last decade, it is now possible to perform satisfactory one stage procedures for a variety of esophageal lesions including congenital atresia with tracheoesophageal fistula, acquired tracheoesophageal fistula, acquired stricture, perforation or rupture of the esophagus, diverticulum, cardiospasm, benign tumor, and carcinoma.

REFERENCES

1. Adams, H. D.: Diverticula of Thoracic Esophagus, J. Thoracic Surg. *17*:639-645 (Oct.) 1949.

2. Adams, W. E., and Phemister, D. B.: Carcinoma of Lower Thoracic Esophagus; Report of Successful Resection and Esophagogastrostomy, J. Thoracic Surg. *7*:621-632 (Aug.) 1938.

3. Harrison, A. W.: Transthoracic Small Bowel Substitution in High Stricture of Esophagus, J. Thoracic Surg. *18*:316-326 (June) 1949.

4. Lynch, J. P.: Spontaneous Perforation of Esophagus, New England J. Med. *241*:395-400 (Sept. 15) 1949.

5. Pack, G. T.: Introduction to Symposium on Cancer of Esophagus and Gastric Cardia, Surgery *23*:867-873 (June) 1948.

6. Sweet, R. H.: Treatment of Carcinoma of Esophagus and Cardiac End of Stomach by Surgical Extirpation; 203 Cases of Resection, Surgery *23*:952-975 (June) 1948.

7. Wookey, H.: Surgical Treatment of Carcinoma of Pharynx and Upper Esophagus, Surg., Gynec. & Obst. *75*:499-506 (Oct.) 1942.

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ABSTRACT OF DISCUSSION

DR. A. W. HARRISON, Galveston: In our modern way of taking everything for granted, we rarely stop to appreciate how extensive are the changes that have taken place in a few years. Let me emphasize, as Dr. Paulson has already pointed out, that most of these developments have come about in hardly more than a decade. Nor has progress slackened.

If I may single out a detail rather than being concerned with the whole, I should like to illustrate one or two of the ramifications this development is taking.

As Dr. Paulson indicated, the fundus of the stomach can readily be brought into the neck and, in fact, right up to the pharynx. A malignant lesion of the cervical esophagus can

be excised and the esophagus anastomosed well above the level of the clavicles by a right thoracic approach. At Galveston we believe that lesions of the upper two-thirds of the esophagus can best be attacked in this way in that the cancer can be more readily and adequately dealt with, the procedure is easier on both patient and surgeon, and actual operating time is reduced.

In this procedure the right side of the chest is opened anteriorly and as soon as resectability is determined the abdomen is opened by an assistant. The stomach is mobilized and passed into the chest. While the surgeon concerns himself with the main train of surgery on the esophagus, the abdomen can be closed. In like manner after the esophagus and fundus are brought out a neck incision, the chest can be closed while the anastomosis proceeds in relative ease on the neck. Finally it is dropped back and the neck is closed.

In one case an esophageal cancer was so high that the larynx as well had to be removed by Dr. McReynolds. At the time the preliminary gastrostomy was made a segment of jejunum was isolated and attached as a blind appendage to the fundus of the stomach. In two later stages this appendage was brought up through the chest and anastomosed to the stump of the pharynx so that the patient could eat. We also hope to get him some sort of artificial larynx to compensate as much as possible for the loss of both esophagus and larynx.

AUREOMYCIN THERAPY IN BACTERIAL INFECTIONS RESISTANT TO PENICILLIN AND STREPTOMYCIN

ELLARD M. YOW, M.D., Houston, Texas

WHILE remarkable advances have been made with the use of penicillin and streptomycin in the treatment of infectious diseases, there are a number of problems associated with the administration of these antimicrobial agents. For example, the physician not infrequently encounters bacterial infections resistant to one or both drugs. Approximately 12 per cent of strains of staphylococci are naturally resistant to penicillin[17]; and in surgical wards where penicillin is frequently used in the treatment of infected wounds, as high as 60 per cent of the strains of staphylococci cultured may be resistant.[10] Fortunately, penicillin-resistant strains of pneumococci, gonococci, meningococci, and group A hemolytic streptococci are rarely, if ever, encountered clinically,[4, 9, 11] but resistant strains of nonhemolytic streptococci may be found in some cases of subacute bacterial endocarditis.[2, 11, 12] Some of these strains resistant to penicillin may be sensitive to streptomycin, but there is also a wide variation in susceptibility among different strains of gram-positive and gram-negative bacteria usually sensitive to this drug.[4]

Resistance to these antibiotic agents may develop during therapy. Although this is rarely observed clinically during the administration of penicillin,[8] the development of resistance of bacteria to streptomycin occurs frequently and progresses rapidly. It has been observed that infections of the urinary tract usually must be controlled within a few days or they will become resistant to the maximum tolerated dose of streptomycin.[6] Approximately 80 per cent of the strains of Mycobacterium tuberculosis cultured four months after beginning streptomycin therapy are found in vitro to be streptomycin-resistant.[15]

From the Department of Internal Medicine, Baylor University College of Medicine.

Read before the Section on Medicine, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

Severe toxic reactions may result from streptomycin therapy, depending upon the size of the dose and the duration of therapy.[18] The most frequent of these is damage to the eighth cranial nerve, though this problem has been somewhat less important with the use of the reduced salt of streptomycin, dihydrostreptomycin.[7] Allergic reactions to penicillin occur not infrequently and may be severe enough to necessitate its discontinuance or to prevent its use in subsequent infections.[13, 14]

Neither of these antibiotics has been effective in a large group of bacterial diseases. These include those caused by the salmonella and proteus group of organisms and many strains of Aerobacter aerogenes, Pseudomonas aeruginosa, Escherichia coli, and Streptococcus faecalis.

The problem of therapy in these diseases and the disadvantages associated with the administration of penicillin and streptomycin prompted the laboratory and clinical investigation of the effect of aureomycin in a group of patients with infections of known etiology failing to respond to these agents. In each case the causative organism was isolated after the infection had failed to respond to sulfonamide, penicillin, and streptomycin therapy. In a few instances, these were included in the study cases in which only two of these agents were administered because of a history of hypersensitivity to the other. In a case of typhoid fever neither of these drugs was administered prior to aureomycin therapy because of their previously observed uniform failure in the treatment of this disease. The causative organisms were tested in vitro for sensitivity to penicillin, streptomycin, and aureomycin and the patient was then given a therapeutic trial on aureomycin.

The results of aureomycin therapy in this group of patients are summarized in tables 1 through 6, and

the results of the in vitro sensitivity tests are compared to the clinical response to therapy.

DISCUSSION

It is of particular interest that aureomycin was effective in 5 cases of staphylococcic bacteremia that had failed to respond satisfactorily to sulfadiazine, penicillin, and streptomycin. The mortality rate of untreated staphylococcic bacteremia ranges between 75 and 80 per cent. Since the introduction of the sulfonamides and penicillin, the mortality rate has been reduced to less than 20 per cent. Experience with aureomycin thus far would indicate that it is probably the drug of choice in severe staphylococcal infections.

Recurrent urinary infections have often presented difficult problems in therapy, since they are often due to such notoriously resistant organisms as Pseudomonas aeruginosa, Proteus vulgaris, Aerobacter aerogenes, and Streptococcus faecalis. Eleven patients with infections of the urinary tract due to Aerobacter aerogenes studied in this group responded favorably to aureomycin therapy. Four patients with urinary infections due to Escherichia coli and 1 with infections due to Micrococcus pyogenes variant aureus responded promptly to the administration of aureomycin. Five patients with infections due to Proteus and 3 with infections caused by Pseudomonas aeruginosa failed to improve when given aureomycin.

The response to aureomycin therapy of the mixed urinary infections depended on the sensitivity of the strains of bacteria taking part in the infection. In those cases in which either Proteus or Pseudomonas aeruginosa was present, the response to the administration of aureomycin was unfavorable. The recurrence of infection following therapy was largely governed by the underlying anatomic changes in the urinary tract. In those cases in which adequate urinary drainage was not established, subsequent infections with the same or different organisms occurred.

TABLE 1.—*Response of Staphylococcal (Micrococcus pyogenes variant aureus) Infections to Aureomycin.*

Age of Patient	Diagnosis	In Vitro Sensitivity* P units/ml.	S mcg./ml.	A mcg./ml.	Previous Therapy*	Aureomycin Therapy Dosage	Duration (days)	Results
3 yr.	Acute osteomyelitis with bacteremia	312	50,000	0.6	P, S, Su	100 mg. every 6 hr.	30	Recovery
14 yr.	Acute osteomyelitis with bacteremia	3,125	40	1.0	P, S	300 mg. every 8 hr.	21	Recovery
11 mo.	Osteomyelitis of skull with brain abscess	312	50,000	0.8	P, S, Su	50 mg. every 4 hr. 100 mg. every 4 hr.	7 14	Recovery
17 mo.	Agranulocytosis with bacteremia	>50	>50	0.6	P, S, Su	50 mg. every 4 hr.	14	Temporary improvement; fatal Pseudomonas bacteremia
62 yr.	Diabetes with bacteremia	100	10,000	1.0	P, S, Su	500 mg. every 6 hr.	7	Recovery
5 mo.	Chronic interstitial pneumonitis	50	3.2	0.6	P, S, Su	50 mg. every 4 hr.	14	Slight improvement
28 yr.	Chronic conjunctivitis	10,000	10,000	0.8	P, Su	Local instillation	14	Recovery
50 yr.	Multiple metastatic abscesses	10,000	10	0.8	P, S, Su	59 mg. intramuscularly every 6 hr.	14	Slight improvement
11 mo.	Chronic otitis media	50	6.25	0.8	P, Su	100 mg. every 8 hr.	12	Recovery
60 yr.	Urinary infection	1,000	100	1.56	P, S, Su	500 mg. every 6 hr.	6	Recovery

*P, penicillin; S, streptomycin; Su, sulfonamide; A, aureomycin.

TABLE 2.—*Response of Urinary Infections Due to Aerobacter aerogenes to Aureomycin.*

Age of Patient	In Vitro Sensitivity* P units/ml.	S mcg./ml.	A mcg./ml.	Aureomycin Therapy† Dosage	Duration (days)	Results
8 mo.	>5,000	>5,000	1.0	50 mg. every 4 hr.	12	Recovery
18 yr.	>2,500	>25,000	4.0	500 mg. every 6 hr.	10	Recovery
78 yr.	>10,000	>10,000	6.25	500 mg. every 6 hr.	7	Recovery
80 yr.	>10,000	>10,000	6.25	500 mg. every 6 hr.	7	Recovery; subsequent reinfections with Proteus and Pseudomonas
18 yr.	>10,000	>10,000	6.25	500 mg. every 6 hr. 250 mg. every 6 hr.	4 2	Recovery; subsequent reinfections with Proteus and Pseudomonas
68 yr.	>10,000	>10,000	6.25	500 mg. every 6 hr.	7	Recovery; subsequent reinfections with Proteus and Pseudomonas
57 yr.	>10,000	>10,000	6.25	500 mg. every 6 hr.	7	Recovery
24 yr.	>10,000	>10,000	3.12	500 mg. every 6 hr.	7	Recovery
13 mo.	>10,000	>10,000	3.12	50 mg. every 6 hr.	7	Recovery
36 yr.	>10,000	>10,000	6.25	500 mg. every 6 hr.	7	Recovery
60 yr.	>10,000	>10,000	3.12	500 mg. every 6 hr.	7	Recovery

*P, penicillin; S, streptomycin; A, aureomycin.
†All patients had been treated previously with penicillin, streptomycin, and sulfonamides.

One case of puerperal sepsis with a bacteremia due to Escherichia coli responded dramatically to aureomycin therapy after failing to improve with the administration of large doses of sulfonamides, penicillin, and streptomycin. A case of meningitis due to Pseudomonas aeruginosa failed to respond to aureomycin when given over a two-week period.

The clinical response to aureomycin therapy closely paralleled the in vitro sensitivity of the causative organisms except in the cases of bacterial endocarditis and typhoid fever. Only Pseudomonas aeruginosa and the proteus group of bacteria were found to be resistant in vitro to aureomycin in concentrations of 12.5 mg. per milliliter or greater. These organisms were not eliminated from the infections studied in this group by the administration of aureomycin. The reason for the failure of aureomycin therapy in 2 cases of subacute bacterial endocarditis due to aureomycin-sensitive strains of Streptococcus viridans is unknown. It is extremely difficult to explain the failure of aureomycin therapy in typhoid fever when chloramphenicol, which is closely related in its antibacterial activity, has been found to be effective.

The clinical and bacteriologic response of patients to aureomycin therapy was often not as prompt as that seen with the administration of penicillin or streptomycin. In serious infections the clinical response was not evident in some cases for forty-eight to seventy-two hours after beginning aureomycin therapy. The most likely explanation for this difference in response is that aureomycin is bacteriostatic in its action, while penicillin and streptomycin are bactericidal. This difference in the action of aureomycin probably also explains the clinical observation of relapses following the cessation of therapy in some cases when the drug has been maintained for a short period of time and discontinued before the patient has developed sufficient immunity to control the infection.

Aureomycin was administered orally in this group of patients in doses ranging from 10 to 60 mg. per kilogram of body weight per twenty-four hours. The usual dose was 30 mg. per kilogram per twenty-four hours, or in an adult of average weight, 500 mg. given every six hours.

Aureomycin hydrochloride was given intravenously in some patients in whom oral administration was impossible. The dose in adults ranged from 500 to 2,000 mg. per day, given as a 0.05 to 0.1 per cent solution in saline or glucose and water. A chemical thrombophlebitis occurred in 1 patient subjected to this method of administration. Because of the severe local irritation, resulting in sterile abscesses in some cases, intramuscular aureomycin was discontinued.

Severe toxic reactions from aureomycin therapy were not observed, but minor unpleasant side reactions were not uncommon. The most frequent side reaction noted in this group of patients and others treated with aureomycin in this clinic was epigastric distress, and the most serious reaction was vomiting. These symptoms are apparently due to the irritating effect of the acidity of the drug rather than to any central effect, but they have occasionally been observed with the parenteral administration of large doses of aureomycin. The gastrointestinal symptoms were greatly diminished by administering the drug with meals or with a glass of milk. The use of alkalies in conjunction with aureomycin is not recommended at present because of the rapid loss of activity of the drug in vitro in an alkaline solution. The upper gastrointestinal tract symptoms were frequently decreased by the simultaneous administration of acid absorbants, but these substances have been shown to decrease significantly the level of aureomycin in the

TABLE 3.—*Response of Infections Due to Escherichia coli to Aureomycin.*

Age of Patient	Diagnosis	In Vitro Sensitivity* P units/ml.	S mcg./ml.	A mcg./ml.	Previous Therapy*	Aureomycin Therapy Dosage	Duration (days)	Results
32 yr.	Puerperal sepsis with bacteremia	>10,000	>10,000	3.12	P, S, Su	1.0 Gm. every 4 hr. 0.5 Gm. every 4 hr.	4 7	Recovery
36 yr.	Urinary infection	>10,000	>10,000	3.12	P, S, Su	500 mg. every 6 hr.	7	Recovery
60 yr.	Urinary infection	>10,000	>10,000	6.25	P, S, Su	500 mg. every 6 hr.	7	Recovery
22 mo.	Urinary infection	>10,000	>10,000	3.12	P, S, Su	100 mg. every 8 hr.	7	Recovery
48 yr.	Urinary infection	1,000	10	6.25	P, Su	500 mg. every 6 hr.	7	Recovery

*P, penicillin; S, streptomycin; Su, sulfonamide; A, aureomycin.

TABLE 4.—*Response of Proteus Infections in the Urinary Tract to Aureomycin.*

Age of Patient (yr.)	In Vitro Sensitivity* P units/ml.	S mcg./ml.	A mcg./ml.	Aureomycin Therapy† Dosage	Duration (days)	Results
39	>10,000	>10,000	25.0	500 mg. every 6 hr.	7	No improvement
76	>10,000	>10,000	100	500 mg. every 6 hr.	7	No improvement
64	>10,000	>10,000	100	500 mg. every 6 hr.	7	No improvement
68	>10,000	>10,000	25.0	500 mg. every 6 hr.	7	No improvement
30	>10,000	>10,000	50.0	500 mg. every 6 hr.	7	No improvement

*P, penicillin; S, streptomycin; A, aureomycin.
†All patients had been treated previously with penicillin, streptomycin, and sulfonamides.

blood. In general, the nausea and vomiting were related to the size of the dose and the total amount of aureomycin given in a day. When more than 2 Gm. were given in a twenty-four hour period, the symptoms were frequently observed. The nausea and vomiting could be controlled in most cases by omitting a dose of aureomycin, by temporarily decreasing the dose, or by giving smaller amounts at more frequent intervals.

Other untoward symptoms noted accompanying aureomycin therapy include frequent, loose stools and burning and itching of the rectum with defecation. Some patients complained of a mild diuretic effect, and transient dizziness was noted in 1 case. Exacerbations of monilial vaginitis were not infrequently noted, and glossitis indistinguishable clinically from that due to niacin deficiency was seen in 2 cases. Two patients during aureomycin therapy developed ulcerative mucosal lesions similar to those seen in herpetic stomatitis. Evidence of hypersensitivity to the drug was not observed.

The development of bacterial resistance to aureomycin was not noted in this group of patients. In those cases in which relapse followed aureomycin therapy, the bacteria recovered had not changed in their sensitivity to the antibiotic.

TABLE 5.—*Response of Pseudomonas Aeruginosa Infections to Aureomycin.*

		In Vitro Sensitivity*			Aureomycin Therapy†		
Age of Patient	Diagnosis	P units/ml.	S mcg./ml.	A mcg./ml.	Dosage	Duration (days)	Results
18 yr.	Meningitis	>10,000	>10,000	50	500 mg. every 6 hr.	14	No improvement
48 yr.	Pneumonia	>10,000	>10,000	62.5	500 mg. every 6 hr.	10	No improvement
17 mo.	Bacteremia	>10,000	>10,000	62.5	50 mg. every 6 hr.	14	No improvement
68 yr.	Urinary infection	>10,000	>10,000	25.0	500 mg. every 6 hr.	7	No improvement
30 yr.	Urinary infection	>10,000	>10,000	50	500 mg. every 6 hr.	7	No improvement
57 yr.	Urinary infection	>10,000	>10,000	50	500 mg. every 6 hr.	5	No improvement

*P, penicillin; S, streptomycin; A, aureomycin.
†All patients had been treated previously with penicillin, streptomycin, and sulfonamides.

Aureomycin was administered to patients in whom previous therapy with the sulfonamides, penicillin, and streptomycin had been ineffective. These include cases of staphylococcal infections, 23 urinary tract infections, 1 case of Escherichia coli bacteremia, 1 case of meningitis due to Pseudomonas aeruginosa, 2 cases of subacute bacterial endocarditis due to Streptococcus viridans, and 1 case of typhoid fever. The response to the antibiotic was favorable except in those infections due to Proteus, Pseudomonas aeruginosa, and in the case of typhoid fever. The cases of bacterial endocarditis relapsed following cessation of therapy. The comparative in vitro sensitivity of the organisms isolated from these patients to penicillin, streptomycin, and aureomycin is tabulated.

The doses used in most cases were 30 mg. per kilogram of body weight per twenty-four hours, or in an adult, 0.5 Gm. every six hours orally.

No serious toxic reactions were observed, though nausea and vomiting and a change in the character of the stools occurred not infrequently. Other untoward reactions observed include burning of the rectum, pruritis ani, monilial vaginitis, glossitis, and stomatitis.

TABLE 6.—*Response of Miscellaneous Bacterial Infections to Aureomycin.*

			In Vitro Sensitivity*				Aureomycin Therapy		
Age of Patients (yr.)	Diagnosis	Bacteriology	P units/ml.	S mcg./ml.	A mcg./ml.	Previous Therapy*	Dosage	Duration (days)	Results
8	Subacute bacterial endocarditis	Streptococcus viridans	6.0	12.0	1.0	P	500 mg. every 6 hr.	45	Temporary improvement
30	Subacute bacterial endocarditis	Streptococcus viridans	1.56	6.25	1.0	P, Su	500 mg. every 6 hr.	60	Temporary improvement
21	Typhoid fever	Salmonella typhosa	50	1.56	6.25	None	500 mg. every 6 hr.	14	No definite effect

*P, penicillin; S, streptomycin; Su, sulfonamide; A, aureomycin.

SUMMARY

The occurrence of natural and acquired resistance among strains of bacteria, toxic and allergic reactions, and the unsatisfactory clinical response of infections due to the salmonella and proteus group of organisms are problems associated with sulfonamide, penicillin, and streptomycin therapy. These problems prompted the clinical investigation of aureomycin in infectious diseases failing to respond to these agents.

REFERENCES

1. Bryer, M. S., and others: Aureomycin; Experimental and Clinical Investigations, J.A.M.A. *138*:117-119 (Sept. 11) 1948.

2. Christie, R. V.: Penicillin in Subacute Bacterial Endocarditis: Report to Medical Research Council on 269 Patients Treated in 14 Centers Appointed by Penicillin Clinical Trials Committee, Brit. M. J. *1*:1-14 (Jan. 3) 1948.

3. Duggar, B. M.: Aureomycin, Product of Continuing Search for New Antibiotics, Ann. New York Acad. Sc. *51*:177-180 (Nov. 30) 1948.

4. Finland, M.: Some Aspects of Uses of Chemotherapy and Antibiotics, Cincinnati J. Med. *29*:317-328 (June) 1948.

5. Finland, M.; Collins, H. S.; and Paine, T. F., Jr.: Aureomycin, New Antibiotic, Results of Laboratory Studies and of Clinical Use in

100 Cases of Bacterial Infections, J.A.M.A. *138*:946-949 (Nov. 27) 1948.

6. Harris, H. W., and others: Streptomycin Treatment of Urinary Tract Infections, with Special Reference to Use of Alkali, Am. J. Med. *2*:229-250 (March) 1947.

7. Hinshaw, H. C.; Feldman, W. H.; Carr, D. T.; and Brown, H. A.: Clinical Administration of Dihydrostreptomycin in Tuberculosis; Preliminary Report, Am. Rev. Tuberc. *58*:525-530 (Nov.) 1948.

8. Langohr, J. L.; Owens, C. R.; and Cope, O.: Bacteriologic Study of Burn Wounds; Comparison of Bacterial Flora of Burn Wounds of Patients Treated with Sulfonamides or Penicillin, Ann. Surg. *125*:452-504 (April) 1947.

9. Miller, C. P.: Development of Bacterial Resistance to Antibiotics, J.A.M.A. *135*:749-751 (Nov. 22) 1947.

10. North, E. A., and Christie, R.: Observations on Sensitivity of Staphylococci to Penicillin, M. J. Australia *2*:44-46 (July 14) 1945.

11. Paine, T. F., Jr.: Problems Relating to Bacterial Resistance, Bull. New England M. Center *10*:181-184, 1948.

12. Schneierson, S. S.: Serological and Biological Characteristics and Penicillin Resistance of Nonhemolytic Streptococci Isolated from Subacute Bacterial Endocarditis, J. Bact. *55*:393-399 (March) 1948.

13. Shaffer, J. O.: Generalized Exfoliative Dermatitis Due to Penicillin, New England J. Med. *238*:660-661 (May 6) 1948.

14. Shawyer, R. A.: Allergy to Penicillin, Brit. M. J. *1*:547 (March 20) 1948.

15. Sixth Streptomycin Conference, Minutes edited by Veterans Administration Branch Office 8, Tuberculosis Section, St. Paul, Minn., Oct. 21-24, 1948, pp. 169-189.

16. Spink, W. W.; Ferris, V.; and Vivino, J. J.: Comparative in Vitro Resistance of Staphylococci to Penicillin and to Sodium Sulfathiazole, Proc. Soc. Exper. Biol. & Med. *55*:207-210 (March) 1944.

17. Spink, W. W.; Braude, A.; Casteneda, M. R.; and Goytia, R. S.: Aureomycin Therapy in Human Brucellosis Due to Brucella Melitensis, J.A.M.A. *138*:1145-1148 (Dec. 18) 1948.

18. Tompsett, R.: Relation of Dosage to Streptomycin Toxicity, Ann. Otol., Rhin. & Laryng. *57*:181-188 (March) 1948.

19. Waisbren, B. A., and Hueckel, J. S.: Reduced Absorption of Aureomycin Caused by Aluminum Hydroxide Gel (Amphojel), Proc. Soc. Exper. Biol. & Med. *73*:73-74 (Jan.) 1950.

ABSTRACT OF DISCUSSION

DR. W. W. BONDURANT, JR., San Antonio: My experience with aureomycin has been much more limited and infinitely more poorly controlled than Dr. Yow reports. My experience is less wide largely because of the unpleasant side reactions in the gastrointestinal tract. These reactions are certainly deterrents to its more widespread use. I welcome Dr. Yow's suggestion relative to their control. It is interesting to see a fairly wide variation in the dosage schedule adopted by Dr. Yow. In certain instances a cure was effected by 300 mg. of aureomycin daily. It has been my experience that urinary tract infections responsive to aureomycin sometimes require no more than 500 mg. daily. Dr. Yow has shown that the drug offers renewed hope to those sufferers from chronic pyelonephritis whose obstruction can be corrected. This no doubt is because of the drug's prolonged excretion in high concentration in the urine.

DEVELOPMENTAL ANOMALIES OF THE EYE

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THE principal lines along which fruitful results have been attained in embryology are (1) normal human and comparative embryology, (2) teratology and fetal pathology, (3) genetics, and (4) experimental embryology.

FIELDS OF RESEARCH

Human and Comparative Embryology.—With a given anomaly it must be decided first until what period of development growth would have appeared normal and secondly, whether the abnormality should be classed as (1) simple arrest of development, (2) pure aberration, or (3) arrest with subsequent aberrant growth.

Mann and others have cautioned against classifying anomalies as atavistic. The statement that the individual in his development climbs his own family tree is apt to be misleading. It is true that shadowy memories of many phylogenetic stages occur in the human embryo, but some of them have had their time relations disconcertingly dislocated; in ontogeny phylogeny is telescoped and blurred.

Strictly speaking, an atavistic structure is one resembling a formation normally present in an ancestrally related species, not merely in a lower vertebrate. Therefore, it would be incorrect to refer to the pigmented glial tissue sometimes found on the optic disk of human beings as atavistic simply because it resembles the conical pecten of the lizard, inasmuch as the two are not ancestrally related. Again, the word ontogeny should not be limited to mean development only until the time of birth. Birth is only an incident occurring in the development of the individual beginning with the zygote and extending to the first evidences of senility. I think that even in senility a suborganogenetic classification should be made, since one organ may still be developing while another is in the throes of degeneration.

Teratology and Fetal Pathology.—Abnormalities can be classified and a certain amount of information gleaned concerning their possible etiology. The individual develops throughout the organogenetic period during which the pluripotential cells, developing into mono-organic structures, lose their pluripotentiality and form a given organ, such as the eye. Although the remainder of ontogeny could be developed into the periods fetal, postnatal, and so forth, they would be merely subdivisions of that period in which an organ develops with concomitant regressions, such as development of the lens and regression of the hyaloid system; therefore, such classification for the purposes of this paper is not essential.

It is necessary, however, to remember that in this organogenetic period the grosser abnormalities are

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formed. This is obvious, since before a tissue becomes histologically differentiated, a noxious agent would cause gross abnormality or death rather than disease, such as leukocytosis, fibrosis, and hypertrophy. Such diseases are possible only after the fetus is formed. A rule, then, is the later the action of a noxious agent, the greater its resemblance to postnatal reaction.

The students of teratology have postulated three main actions of a noxious agent: (1) germinal, (2) transplacental, and (3) mechanical.

Genetics.—Inasmuch as genetics is such a tremendous subject, for the purposes of this paper it can be dismissed except for one consideration. While acceptance of genetic theories provides a method whereby certain occurrences can be predicted with precision and certain difficult occurrences explained, it is difficult after a consideration of mutations and experimental embryology to agree with the idea that predestination lies within the chromosomes of the zygote.

Without a long discussion of transplacental passage of substances which act on the fetus, suffice it to say that toxins, hormones, foodstuffs, drugs, and bacteria pass through the placenta from mother to fetus. They may affect perhaps both and perhaps only the fetus, produce characteristics sometimes similar in each and sometimes widely different, and be conducted sometimes by the amniotic fluid, and sometimes by the blood. Mechanical pressure on growing parts produced by an abnormally adherent amnion or an excess or deficiency of amniotic fluid may result in an anomaly.

Experimental Embryology.—In the field of experimental embryology certain broad principles of development have been stated, which will be defined but not fully considered. At the outset all cells of the morula are totipotential, meaning that if the morula is divided in half, two individuals will be formed, one smaller than the other but perfect. When the chromatin has been sufficiently distributed, the cells are no longer totipotential but pluripotential, that is, capable of differentiating into all the tissues of a given organ but not into all the tissues of the body. At this time the anlage, a patch of cells on the surface of the gastrula, by various outpouchings, invaginations, and differential growth rates comes to resemble in form and position the adult organ. It is only after this stage that histologic differentiation takes place.

Playing an important role throughout this process are the organizers of the first order which can initiate the development of the entire embryo; for example, if the dorsal lip of the blastopore were grafted onto the surface of another blastoderm, it could initiate a second embryo on the body of the first. Organizers of the second grade are those which can initiate the structure of a single organ such as the optic vesicle, which has the power to stimulate any portion of the body surface to form a lens. Also playing an important role are the axial gradients which cause one part of the body or of an organ to develop with greater rapidity than another; an example is the gradient from the dorsal part of the optic cup to the optic fissure and after the closure of the fissure, the gradient from the posterior pole to the anterior lip of the cup.

GROSS ABNORMALITIES

Anencephaly, tower skull, cyclopia, and anterior and posterior dichotomy are conditions which need only be mentioned. Congenital cystic eye and microphthalmia with orbital cyst, however, should be discussed because they are conditions compatible with life occasionally being encountered and requiring differentiation in a clinical practice.

Anophthalmos will result if there is complete suppression of the optic outgrowth. If, however, development proceeds to optic outgrowth but ceases before invagination, the condition of congenital cystic eye results. The eye is absent and its place is taken by a large, thin walled cyst with normal eyelids. The cyst is centrally placed or even a little upward. Histologically, as would be expected, the cyst wall is made of a fibrous tissue representing the sclera into which are attached the muscles of the orbit. Posteriorly there is a layer of pigmented cells and anteriorly, neural tissue which is poorly differentiated but obviously preretinal. No pupil, iris, or vitreous cavity is present, but there may be a small, ill-defined, degenerate lens outside the cyst wall.

If the cyst is deep to the lower lid which bulges before it, microphthalmia with cyst must be considered. In this condition, of course, an eye has been formed; therefore, the organogenetic development of the eye is a little more advanced. The microphthalmic eye may be well developed and the cyst only a small out-pouching which is almost invariably located below the horizontal meridian, or the eye may be so small and the cyst so large that no eye is visible.

The clinical importance of buphthalmos and hydrophthalmos, is sufficient for their consideration, yet their causation is so poorly understood that a dissertation in such a general discussion hardly seems warranted. Suffice it to say that the former is probably due to intrauterine inflammation while the latter is caused by faulty development of mesodermal tissue at the angle of filtration. It is interesting to contemplate the possibility that glaucoma simplex of adults may be of similar origin to hydrophthalmos. If differentiation of the mesodermal tissue of the filtration angle has totally failed, then hydrophthalmos occurs, whereas if differentiation is merely incomplete, it seems possible

that drainage might be maintained until the sclerosing changes of adult life are superimposed.

COLOBOMAS

When a cyst of the eye cannot be diagnosed, there may be various abnormalities, almost invariably colobomas of the iris, ciliary body, and choroid. Colobomas occurring in the fetal ocular cleft running downward and slightly inward around the eye from the center of the optic disk to the pupilary margin slightly to the nasal side of the center of the lower border of the pupil are typical. Similar defects found elsewhere are atypical. Colobomas hardly require clinical description; their etiology and intimate relationship, however, are of interest.

There is no doubt that the cause of coloboma is failure of proper closure of the fetal ocular cleft but whether this failure is primarily caused by persistence of mesoderm in the cleft or is primarily ectodermal in origin is not known. Currently the consensus is that the neural ectoderm is at fault. To understand the actual mechanical occurrence, it must be remembered that the inner wall of the optic cup has a strong tendency to evaginate and that development of the histologic structures from this wall is latest at this point. The question arises whether the choriocapillaris is dependent for its formation on the outer wall of the cup or whether the development of the pigment epithelium is dependent upon the choriocapillaris. Although much discussion has taken place, it is currently thought that the dominant factor is ectoderm and not mesoderm. In any event, at the margin of the fissure a tendency exists for evagination of the inner layer and, depending upon the stage, one or two cysts or a total, bridge, ectatic, or cystic coloboma may be present. It must be remembered that the ectatic coloboma is essentially different from the cystic coloboma, the cavity of the former being the vitreous body and the latter the optic vesicle. Inasmuch as coloboma of the iris is probably of different origin and is so much more frequently atypical, it will be discussed later.

Crescents, colobomas, and crater-like holes of the optic disk; opaque nerve fibers; Leber's disease; retinitis pigmentosa; Oguchi's disease; and amaurotic family idiocy are among a host of anomalies which need only to be mentioned. Of further interest, however, is gliosis of the disk and pseudoneuritis. To understand this condition it is necessary to remember something of the formation of Bergmeister's papilla. The nerve fibers in reaching the optic nerve cut off a small portion of the neuro-epithelium (that is, the inner wall of the cup), isolating these cells, which normally do not differentiate into retina but remain as glia-like elements of ectodermal origin. Variations in depth of the physiologic cup are explained to be the result of excessive or deficient atrophy of this glial tissue. Its excessive development may create a pseudoneuritis or connective tissue veils about the vessels, both of which are commonly seen.

Coloboma of Macula

There are two sets of theories which have been propounded for coloboma of the macula: (1) developmental and (2) pathologic.

We can dispose of the term coloboma in considering the first of the developmental theories, which is that the coloboma is created by a failure of closure of the fetal fissure. It was necessary for proponents of this theory to expound the idea that there normally occurred a rotation of the fetal fissure since it was known to lie inferiorly. The theory does not tolerate scrutiny since it is known that the fissure does not rotate 90 degrees or even 45 degrees. In addition to this, coloboma of the macula has been found in the presence of coloboma of the choroid and retina and therefore could not be created by failure of closure of the same fissure. The early anatomists, having discovered the horizontal raphe, decided that it was the point of closure; their decision, however, is known not to be true. Since a coloboma in the sense they thought of is incorrect, the term can be disposed of and the word metaplasia substituted.

The second developmental theory takes for granted that there is a localized and faulty differentiation of the wall of the optic cup. Whether the formation of pigment is dependent on the choriocapillaris or vice versa is not known, since they appear practically simultaneously in development. The question arises, therefore, whether the primary failure is in the neural ectoderm. Indeed it is not, since retinal vessels may exist in the coloboma and since therefore the retina may have developed normally until at least the fifth or sixth month. The theory does not take into account the deeply pigmented type of coloboma unless it is further assumed that the inner wall of the cup has undergone an abnormal differentiation into pigment-bearing cells.

The third developmental possibility is primary failure of the mesoderm to develop normally with secondary failure of the ectoderm. It has been contended that since pigment appears to be related to the choriocapillaris, if the latter failed to develop over a given area, pigment would be absent in the outer wall of the optic cup over the same area and a white patch would result. There is no evidence, however, that contact with pigmented epithelium is necessary to the normal development of the inner wall of the optic cup; therefore, the associated failure of the retinal layer in macular dysplasia is not explained.

Neither is the deeply pigmented type of dysplasia elucidated by this theory.

Of the pathologic theories, the hemorrhagic or infantile choroiditis theories need not be discussed. The third theory offers the most interesting speculation and accounts for the three types of coloboma. It already has been indicated in this paper that the earlier an infection occurs, the less do its results resemble the inflammatory reactions of adult tissues. Thus, if the process starts extremely early, that is, the first month, the growth of the embryo is probably so fundamentally interfered with that it becomes incapable of developing at all. If infection occurs during the major part of the organogenetic period up to 3 months, the usual inflammatory reaction does not occur but growth may be retarded and perverted. The later it occurs, the more nearly will infection resemble processes well recognized in post-natal life. Since the well known selective action of certain infective processes on the adult macula cannot be denied, there seems no reason to suppose that the fetal macula is any more immune; indeed, being the most highly specialized and the latest developed region of the retina, it is probably the first to succumb.

The three etiologic types of coloboma and the gradations between them can be brought into line under this third pathologic heading, a maneuver which is impossible with any other single theory. The pigmented type probably occurs latest in fetal life, during the eighth or ninth month. The nonpigmented type probably occurs during the fifth or sixth month; at this time the retinal vessels have apparently existed since they stop abruptly at the coloboma. The third type associated with abnormality of vessels is interesting and also explainable. Vessels coming through the coloboma from the outside of the globe are probably ciliary vessels which have penetrated to the retina and become cilioretinal after Bruch's membrane was destroyed. Those vessels which apparently come from the coloboma and enter the vitreous are probably the result of an abnormal adhesion of the vasa hyaloidea and retina at the time of the inflammation.

IRIS DEFECTS

Earlier in this paper little was said of the iris, a bare mention being made of its relation to typical coloboma of the retina and choroid. The first consideration in regard to the iris should be aniridia, which properly should be termed clinical aniridia since a small rim of iris is always apparent when the eye is examined under the microscope. Although not demonstrable until the latter part of fetal life, the defect is obviously germinal since the iris is formed by the forward growth of the margin of the cup at the extreme end of the organogenetic period. The question immediately arises whether the failure is primarily ectodermal or mesodermal. The probabilities of the mesoderm being at fault are that if the mesodermal wedge creating the iris stroma, which constitutes the framework, does not form, it follows that the more important structures will not form either. This theory, however, does not account for the almost constant coincidental defects of the macula and the lens.

A second mesodermal theory is that a failure of the neural ectoderm is brought about by undue persistence of mesoderm, especially the capsulopupillary portion of the tunica vasculosa lentis. This theory is attractive since the tunica vasculosa lentis undoubtedly plays an important role in the production of coloboma of the iris. The theory, however, presupposes that the mesodermal iris would share in the atrophy of the entire tunica vasculosa lentis; there is no reason to believe this supposition is true. It is also difficult to believe, from what is known of genic factors, that atrophy should be delayed just long enough to inhibit atrophy of an adjacent part. It seems much more probable that primary failure of one structure should occur with secondary atrophy of another.

The ectodermal theory is the most acceptable one since it seems to explain everything that takes place, including the macular failure and lens abnormalities. According to this theory the arrest in development of the optic cup would take place after the retina is differentiated but before the macula is formed. Lens failures could be explained on the basis of the fact that the margin of the optic cup undoubtedly has a great influence on the formation of the lens. The lens, it will be recalled, is formed by the surface ectoderm; in man, however, the lens is not self-determining but is initiated and controlled by contact with the neural ectoderm. There is no reason to believe that the rim of the optic cup does not influence lens formation for a long time; indeed, there is reason to believe that it does.

The most probable cause of coloboma of the iris is mesodermal persistence with inhibition of the ectoderm.

Polycoria, iris diastasis, iris dehiscence, microcoria, ectropion uvae congenitum, persistence of pupillary membrane, heterochromia, heterochromic iritis, fetal iritis, albinism, and cysts of the iris cannot properly be so summarily considered. For the purposes of this paper, however, it may be stated that the philogenetic history of the iris is one of instability. Therefore, the ontogenetic development should be anticipated to be similar—and such is the case.

CATARACTS

Primary and secondary congenital aphakia, posterior lenticonus, anterior lenticonus, lentiglobus, and um-

bilication of the lens are of interest but not as interesting as developmental cataract. Any opacity of the lens, whether total or microscopic in size, is technically a cataract; all cataracts are aberrations and never arrests in development. They may be classified according to appearance or position but neither classification is clinically satisfactory. It is known that toxic substances, germinal factors, and disturbances of metabolism may cause cataracts and that senile degeneration of the lens is often hereditary and familial, but as a general rule, the cause of the aberration is a mystery.

Vogt's anterior axial embryonic cataract occurs in the central dark interval nearer the anterior Y than the posterior one. The cataract could be the result of nonabsorption of the cells of the epitrichial layer of the embryonic skin from the developing lens vesicle, but much more probably such cataracts are produced by the nuclei of the lens fibers themselves. A sutural cataract may be a minute amount of cement substance normally present between the ends of the lens fibers in the suture lines in excess which is abnormal in consistency and chemical composition. The architecture of coralliform, fusiform, and axial cataracts can be explained by the histologic study of the normal developmental progress of lens fibers.

Disk shaped cataract has as its most apt embryologic explanation a failure of the nucleus to develop and a growth of the outer fibers, which are plastered to the equatorial region. This type of cataract is germinal and dominant, probably the result of a localized metabolic disturbance, and otherwise is known as Coppock cataract, being named for the family in which the most cases have been seen.

Congenital morgagnian cataract is most likely a denegeration of the outermost zones with preservation of the nucleus. It is probably metabolic and occurs near the end of fetal life.

Lamellar or zonular cataract is associated with a disorder of the whole individual, possibly an endocrine disturbance, with alteration in lens metabolism. Postnatal developmental cataracts are acquired developmental states associated with pathologic conditions presumably caused by endocrine imbalance, that is, mongolism, cretinism, and myotonia atrophica and those opacities unassociated with another defect, that is, coronary, blue dot, and floriform cataracts.

An anterior polar cataract may be the result of the reaction of a previously normal capsule to pressure or irritation or may be a developmental anomaly connected with the lens stalk. A posterior polar cataract is invariably connected with abnormal persistence of the hyaloid artery.

Coloboma of the lens is primarily a defect of the zonule which is associated with or caused by persistence of the capsulopupillary vessels and which appears at three to four months. Ectopia lentis is undoubtedly caused by an abnormal underdevelopment of the zonule.

There are many other interesting developmental abnormalities of the eye, such as cryptophthalmia, but since those already mentioned have been discussed far too briefly, it would seem inadvisable to discuss others with the even greater brevity which would be required.

SUMMARY

Research in the fields of normal human and comparative embryology, teratology and fetal pathology, genetics, and experimental embryology are discussed. The etiology of eye anomalies such as colobomas and cataracts are considered.

812 Fisk Building.

ABSTRACT OF DISCUSSION

DR. THOMAS J. VANZANT, Houston: I do not choose to raise an issue with Dr. Murphy; few ophthalmologists have the depth of understanding of embryology shown in this paper. Normal development is so complicated and difficult to follow that few of them have the temerity to explore the pathology of development.

Certain developmental anomalies are obviously the result of a noxious agent, such as cataracts resulting from German measles. Some anomalies can be recognized as arrests or failures in the proper sequence of normal development, such as typical colobomas. Atypical colobomas, as of the macula, appear to be inflammatory rather than developmental. Heredity unquestionably is involved in other anomalies. In a family of patients whom I have observed, three siblings with an 18 year age spread have identical total color blindness, nystagmus, nyctalopia, and amblyopia. No similar conditions were discoverable in the family tree on either side for at least three generations.

Correct interpretation of anomalies is an important influence in our discussions with the patient and family, particularly in anticipating anomalies in persons yet to be or not to be conceived.

Research Fellowships in Medicine

The American College of Physicians has announced that a limited number of fellowships in medicine will be available from July 1, 1951, to June 30, 1952. Designed to provide an opportunity for research training either in the basic medical sciences or in the application of these sciences to clinical investigation, the fellowships are for the benefit of physicians who are in the early stages of their preparation for a teaching and investigative career in internal medicine.

Assurance must be provided that the applicant will be acceptable in the laboratory or clinic of his choice and that he will be provided with the facilities necessary for the proper pursuit of his work. The stipend will be $2,200 to $3,200. Application forms will be supplied on request to the American College of Physicians, 4200 Pine Street, Philadelphia 4, and must be submitted in duplicate not later than October 1, 1950.

CASE REPORTS

WELL-WATER METHEMOGLOBINEMIA

Report of Two Cases

M. C. CARLISLE, M.D., Waco, Texas

A PHYSICIAN who does rural practice or sees rural patients should be acquainted with the fact that well water having a high nitrate content may cause cyanosis in infants. This is due to the development of methemoglobinemia.

The first case of methemoglobinemia reported in the literature was by Schwartz and Rector in 1940. It occurred in a 2 weeks old infant who lived in the country. The baby's formula consisted of a dry milk and well water. Unfortunately a nitrate estimation was not done on the water.

Methemoglobin is a hydroxide of hemoglobin and is not toxic. Paul and Kemp recently demonstrated that it may be present in the blood normally to the extent of 1 per cent. Greenberg, Lester, and Haggard found that in vitro one molecule of a nitrate ion reacts with two molecules of hemoglobin to form methemoglobin. Methemoglobin does harm by reducing the functioning hemoglobin and thereby deprives the body of oxygen.

In 1905 van den Bergh observed that nitrates in the intestines could be converted to nitrites and that an inflamed mucosa would hasten their absorption. Later, Zobell proved that in vitro organisms such as Escherichia coli, Streptococcus viridans, and pneumococci found in the gastrointestinal tract could convert nitrates to nitrites.

There are many shallow wells in Texas that have a high nitrate content. The intake of this water during infancy might lead to cyanosis. There seems to be a difference of opinion as to the safe nitrate concentration of well water for the preparation of infant's formula. Comly, who was the first investigator to call attention to this syndrome, stated that well water used in infant feeding should possess a nitrate content no higher than 10, or at the most, 20 parts per million. Actually, whether or not cyanosis develops depends on the amount of total nitrate taken in and certain factors favoring its absorption. During the process of digestion nitrates are produced in the intestinal tract, then converted to nitrites, and finally eliminated as ammonia. It was brought out by Ferrant that a diarrhea which would hasten the passage of the intestinal contents might interfere with the conversion of nitrites to ammonia. Any inflammatory condition of the mucosa would accelerate the absorption of the nitrites.

The question naturally arises as to why older children and adults do not develop methemoglobinemia from drinking well water with a high nitrate content. Cornblath and Hartman proved that young infants are more likely to develop methemoglobinemia from the intake of well water with a high nitrate content because of the gastric hypoacidity frequently present in the young infant. This lack of acidity allows the nitrite forming bacteria usually found in the colon to thrive. Not only is this condition present in the neonatal period but it is also present in premature and weak infants. With the infant, therefore, there is a greater amount of nitrite available for absorption and the subsequent development of methemoglobinemia. In older children and adults, because of the more acid gastric juices, nitrates can be assimilated before they are subjected to the reducing action of bacteria.

It is definitely known that an infant weighing from 8 to 10 pounds will use at least six or eight times more water in proportion to body weight than an adult of 150 pounds. Since the infant is more susceptible to diarrhea, which results in congestion and inflammation of the intestinal mucosa, absorption of nitrites as such is facilitated. Although his work has not been published, Lecks is of the opinion that nitrites form methemoglobin more quickly with fetal hemoglobin than adult hemoglobin.

SURVEY OF TEXAS WELLS

In a survey made in cooperation with the State Department of Public Health, 458 samples of water from 185 counties (73 per cent of the total number

TABLE 1.—*Nitrate Content in 458 Samples of Water from Texas Wells.*

Nitrate (parts per million)	Samples No.	Samples %
0-9	246	70
10-19	23	6.5
20-49	64	18.5
50-99	8	2.5
100 and more	9	2.5

Read before the Section on Pediatrics, State Medical Association of Texas, Annual Session, Fort Worth, May 3, 1950.

of counties in Texas) were studied. For the most part these water samples were taken from private, shallow wells, but occasionally lakes and rivers were sampled. Not included in this study were 1,736 samples of water from municipal wells. The samples of water from shallow wells were grouped according to the nitrate content as noted in table 1.

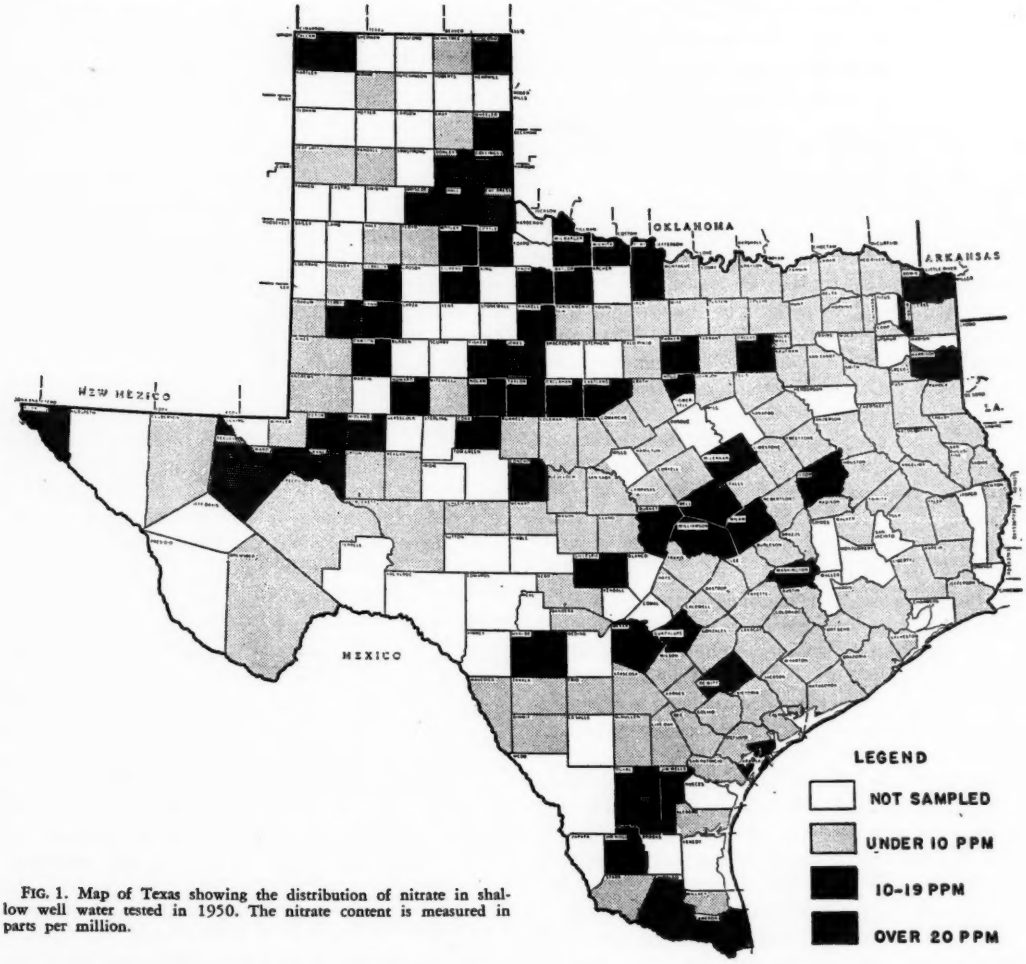

FIG. 1. Map of Texas showing the distribution of nitrate in shallow well water tested in 1950. The nitrate content is measured in parts per million.

Out of the 185 counties studied, 56 or 30 per cent had wells from which the water had over 10 parts of nitrate per million. Nineteen counties had 2 or more wells with the upper limit of nitrate nitrogen. One of these counties had 11 wells which were considered unsafe for the preparation of infant's formula and 3 other counties had 5 or more wells in the same category.

The group of wells having from 10 to 19 parts of nitrate per million had an average depth of 393 feet; those containing 20 to 49 parts per million had an average depth of 221 feet; those with 50 to 99 parts per million had an average depth of 49 feet; and those having more than 100 parts per million had an average depth of 55 feet. It seems definite that the shallow well concerns us most.

The highest nitrate content found in any of the Texas wells was 750 parts per million, and the next highest was 620. This water was used in the preparation of the formula in case 1. Unfortunately bacteriologic studies were not made on all of the samples of water collected. The coliform organisms were found in a few, but for statistical purposes not enough tests were done to draw any conclusions.

The physical characteristics of these wells were similar to those in other states. For the most part they were shallow, ranging from 40 to 100 feet in depth; they were old and had poor casings or none at all. In a few of the wells the casings had been penetrated

by roots or they had partially collapsed because of age and poor construction. Many of the wells were improperly covered, allowing surface water to run or seep in. Some of them were located near barnyards and privies from which they received a seepage of animal excreta and organic waste.

INCIDENCE

Thus far some 41 cases of well-water methemoglobinemia have been reported in the literature. All of these are not proved cases, but most of the patients had been drinking water with a high nitrate content. To my knowledge, the cases which follow are the first reported in Texas. Iowa, in which the condition was first described, has had 21 cases; Canada 6; Kansas and Illinois 3 each; Belgium and Michigan 2 each; and New York, Pennsylvania, Kentucky, Virginia, and Nebraska 1 each. This certainly does not represent the number of cases which have occurred because often the cyanosis is considered to be due to other causes.

CASE REPORTS

CASE 1.—D. S., a white boy, was born normally after an uneventful pregnancy, the third child of healthy parents. The birth weight was 7½ pounds. There was no trouble during the neonatal period. The baby was not given breast milk but was on a formula of evaporated milk 7 ounces, boiled water 15 ounces, and Dextrimaltose No. 1, 3 tablespoons. He took from 3 to 3.5 ounces every three hours.

The infant did well until he was 2 weeks of age, at which time he developed cyanosis. He was admitted to a local hospital where oxygen was given without improvement. In spite of the cyanosis, feedings were taken well and the patient continued to gain weight. The cyanosis subsided after two days in the hospital, and he was allowed to return home.

A few days after the patient returned home the cyanosis again reappeared. At the age of 5 weeks he had a roentgen-ray treatment to his thymus gland; this was followed by a second treatment without benefit. All during this time he was afebrile and except for his color was considered to be doing well.

The boy was admitted to Hillcrest Hospital at the age of 42 days because of the persistence of cyanosis.

Physical examination at this time revealed a well developed and nourished baby weighing 9 pounds 6 ounces. He had a bluish-gray color. The examination failed to reveal any abnormality. The respiratory rate varied from 40 to 50 respirations per minute. The heart was normal; the rate was 136 beats per minute. The infant was given oxygen by use of the Burgess oxygen box but with little or no relief.

It was suspected when he entered the hospital that the well water used in the preparation of his formula might be at fault, so the nurse was instructed to make the new formula with distilled water leaving all the other ingredients the same.

When blood was drawn for methemoglobin and hemaglobin determinations, it was chocolate brown. The methemoglobin was 3.5 Gm. per 100 cc. of whole blood. The hemoglobin concentration was 9 Gm. or 58 per cent. This left only 4.5 Gm. of functioning hemoglobin per 100 cc. of whole blood. By the end of the second day the cyanosis was much improved.

Further laboratory work showed the urine to be normal. The red blood cell count was 3,000,000 per cubic millimeter. The white blood count was 28,000 per cubic millimeter. The differential white blood cell count was normal. The Rh factor was positive on the patient and mother.

Roentgenogram of the chest showed the lung fields clear; the heart was well within normal limits for size, and it showed a normal contour. The thymus was small. There was no evidence of tumors in the mediastinum.

On the third day in the hospital the patient's color was still better, but because of the anemia a transfusion of 100 cc. of citrated blood was given. By this time it seemed apparent that the condition was a well-water methemoglobinemia inasmuch as the only change which had been made was the use of distilled water in the formula. By the end of the week the cyanosis had completely disappeared and the anemia was relieved. The baby gained 7 ounces in weight and was allowed to go home with instructions to the mother to use a different water in the preparation of the formula.

Water was obtained from the well used by the family and was tested by the Bureau of Laboratories, Texas State Department of Health. The water was found to contain 620 parts of nitrate nitrogen per million. A subsequent test was done on this well during the dry season when the water level was low, and it contained 190 parts of nitrate nitrogen per million.

CASE 2.—W. H., a white boy 2 weeks old, was fed on the breast with a complementary feeding of Similac and water. The intake of the extra feeding varied from 2 to 3 ounces after each breast feeding at three hour intervals. The family and prenatal history were not contributory. The boy was the fifth child, was delivered normally, and weighed 8 pounds at birth. The neonatal period was considered normal. The baby was listless the second week of life and slept a lot. When he was 14 days old, he became cyanotic. He was seen the second day of this illness and admitted to Providence Hospital. There was no history of intake of any drug. There was nothing to suggest aniline dye poisoning.

Physical examination revealed a fairly well developed and nourished male infant weighing 8 pounds 12 ounces. The skin and mucous membrane were moderately cyanotic. The fontanel was soft. The heart rate was not especially rapid, and no murmurs were heard. The chest was clear to auscultation and percussion.

Laboratory work showed the urine to be normal. The red blood cell count was 6,100,000 per cubic millimeter with 135 per cent hemoglobin or 21 Gm. The white blood cell count was 19,100 with a normal differential. The baby's blood was type 4, Rh positive. The mother's blood was Rh positive. The blood Wassermann and Kline tests were negative. An analysis of the baby's blood for methemoglobin showed 6 Gm. for each 100 cc. A spinal puncture was done and it revealed 65 mg. of sugar per 100 cc., 75 mg. of protein per 100 cc., and a cell count of 11, all of which were lymphocytes. There were a few crenated red blood cells present.

A roentgenogram of the chest showed the heart and lungs to be normal.

On the second day the cyanosis was much improved. The baby was more alert and he was taking more of his complementary feeding. By the third day the cyanosis was completely gone and the baby was discharged.

Water was obtained from the well used by the family and was tested by the Bureau of Laboratories, Texas State Department of Health. The water was found to have 54 parts of nitrate nitrogen per million.

It is interesting to note that both of these cases

occurred at the age of 2 weeks. One infant was breast fed with a complementary feeding of a powdered milk; the other was fed exclusively on dry milk. Neither patient was troubled with cyanosis while in the hospital. The histories of these two babies are similar to those reported by other authors. Both patients recovered without any specific treatment other than a change of water. This is in agreement with the observation of Halpern and Roche quoted by Ferrant, that if the toxic agent is removed, spontaneous transformation of the methemoglobin into normal hemoglobin takes place and is practically complete after thirty-six hours.

ETIOLOGY AND SYMPTOMS

Ordinarily this condition is found in the infant less than 3 months of age. Usually the baby is fed on a formula of evaporated or powdered milk since this is the type of preparation requiring most water. Rarely is the condition seen in breast fed babies or in infants who are fed undiluted cow's milk mixtures. The water used in preparation of the formula comes from a shallow well having a nitrate content of more than 10 parts per million. It must be remembered that many times the water also contains the coliform organisms. Boiling takes care of the contamination but also concentrates the nitrates.

The mechanism of the production of methemoglobin seems to be that large quantities of nitrates are ingested and as a result of the formation of nitrites and absorption as such methemoglobin is formed. Normally nitrates are reduced to nitrites and then excreted as ammonia as aforementioned.

The symptoms of this condition are cyanosis and sometimes diarrhea. Vomiting may be present. The cyanosis is usually constant but may fluctuate from day to day depending upon the intake of nitrates. This cyanosis is usually not relieved by the administration of oxygen. There is a paucity of physical findings other than a slate gray-blueness. Cyanosis is usually present when methemoglobin exceeds 3 Gm. per 100 cc. of blood. There may be symptoms of anoxia when enough methemoglobin is present to hamper the oxygen carrying capacity of the hemoglobin. These may consist of extreme restlessness or lethargy, air hunger, and even convulsions. Faucett, in his report of twins, had 1 patient survive with a functional hemoglobin as low as 4.3 Gm. per 100 cc. of blood.

DIAGNOSIS

To make a definite diagnosis the physician must have a history of the patient consuming water with high nitrate content. Withdrawal of blood will reveal it to be chocolate brown in color. It should be oxalated or citrated for methemoglobin examination. Mendovy brought out that a presumptive diagnosis can be made even though a blood examination is not done provided the water used in the infant's formula contains more than 10 parts of nitrate per million. If the blood is not examined and the well water is not available for analysis, a presumptive diagnosis can be made by the spontaneous disappearance of the cyanosis following a change of water.

Nitrite determination on the blood may be done. The normal blood nitrite is 10 gammas per 100 cc. At least 10 cc. of blood is needed for this estimation.

There are other sources of nitrates which might cause methemoglobinemia. The use of bismuth subnitrate by the roentgenologists as a contrast medium might cause it. The occasional use of bismuth subnitrate by the physician for the control of diarrhea may be the etiologic agent. Ammonium nitrate used as a diuretic is sometimes at fault. The physician must also take into consideration other factors such as aniline dyes, nitrophenol, sulfanilamide, potassium chlorate, acetanilid, and acetophenetidin U. S. P. (phenacetin) which may cause cyanosis. Perhaps more often, however, congenital heart disease should be considered, especially in those cases in which no murmur is heard and enlargement is not demonstrated. The thymus is often blamed and pulmonary infection as well as diaphragmatic hernia must be ruled out.

PREVENTION AND TREATMENT

Not only should well-water methemoglobinemia be the concern of the practicing physician, but it should also be the responsibility of the State Department of Public Health. The fact that such cases occur is another argument for breast feeding. Whole milk formulas acidified or sweet are safe. Powdered milk in which large quantities of water are used are the most hazardous of all the formulas. Before water from a shallow well is used, it should be tested for nitrates and rejected if it has more than 10 parts per million.

There is a tendency to spontaneous recovery in those cases in which the formula is withheld for twenty-four to forty-eight hours or a change in water is made. On the other hand there is a deepening of the cyanosis if the formula is further diluted, thereby increasing the intake of nitrates. In mild cases from 100 to 200 mg. of ascorbic acid intramuscularly daily has been used. Niacin has also been advocated. The preferred treatment, however, is the giving of methylene blue 1 to 2 mg. per kilogram of body weight intravenously. Brooks has advised that it be given in hypertonic glucose solution because in vitro glucose transforms methemoglobin into oxyhemoglobin. Intramuscular or subcutaneous injection is painful and it frequently causes induration and sometimes necrosis. Hartman and his associates found that methemoglobin caused by sulfanilamide responded well to intravenous methylene blue. They also observed that

65 to 130 mg. of the dye for each kilogram of body weight given by mouth was effective, but they warned against the side effects such as vomiting, diarrhea, tinnitis, and headache. Methylene blue is said to be an effective oxydizing agent and it increases the combustion of the human erythrocyte twenty to thirty times. Ferrant also brought out in his paper that Lederer advises the replacement of methylene blue by thiomine, a violet dye which is not toxic and not methemoglobin formative. It is said to have a reducing power five times greater than that of methylene blue.

Acknowledgment is made of the helpful cooperation, suggestions, and assistance in securing and examining water samples to Drs. George W. Cox, State Health Officer, and Fred P. Helm, Sr., Pediatric and Field Consultant, Maternal and Child Health Division, Texas State Department of Health, Austin.

REFERENCES

1. Brooks, M. M.: What Is Cyanmethemoglobin? Proc. Soc. Exper. Biol. & Med. *32*:1113-1114 (April) 1935.
2. Chapin, F. J.: Methemoglobinemia from Nitrates in Well Water, J. Michigan M. Soc. *46*:938 (Aug.) 1947.
3. Comly, H. H.: Cyanosis in Infants Caused by Nitrates in Well Water, J.A.M.A. *129*:112-126 (Sept. 8) 1945.
4. Cornblath, M., and Hartman, A. F.: Methemoglobinemia in Young Infants, J. Pediat. *33*:421-425 (Oct.) 1948.
5. Faucett, R. L., and Miller, H. C.: Methemoglobinemia Occurring in Infants Fed Milk Diluted with Well Water of High Nitrate Content, J. Pediat. *29*:593-596 (Nov.) 1946.
6. Ferrant, M.: Methemoglobinemia, 2 Cases in Newborn Infants Caused by Nitrates in Well Water, J. Pediat. *29*:585-592 (Nov.) 1946.
7. Goluboff, N.: Methaemoglobinaemia in Infant, Canad. M.A.J. *58*:601-603 (June) 1948.
8. Greenberg, L. A.; Lester, D.; and Haggard, H. W.: Reaction of Hemoglobin with Nitrate, J. Biol. Chem. *151*:665-673 (Dec.) 1943.
9. Harper, E. A.; Cyanosis in Infants from Polluted Well Water, Virginia M. Monthly. *76*:32-34 (Jan.) 1949.
10. Johnson, G. (Iowa City), and others: Nitrate Levels in Water from Rural Iowa, Preliminary Report, J. Iowa M. Soc. *36*:4-7 (Jan.) 1946.
11. Lecks, H. I.: Methemoglobinemia in Infancy, Am. J. Dis. Child. *79*:117-123 (Jan.) 1950.
12. Medovy, H.: Well-Water Methemoglobinemia in Infants; Its Occurrence in Rural Manitoba and Ontario, Journal Lancet *68*:194-196 (May) 1948.
13. Medovy, H.; Guest, W. C.; and Victor, M.: Cyanosis in Infants in Rural Areas (Well-Water Methaemoglobinaemia), Canad. M.A.J. *56*:505-508 (May) 1947.
14. Paul, W. D.; and Kemp, C. R.: Methemoglobin: Normal Constituent of Blood, Proc. Soc. Exper. Biol. & Med. *56*:55-56 (May) 1944.
15. Schwartz, A. S., and Rector, E. J.; Methemoglobinemia of Unknown Origin in 2 Week Old Infant, Am. J. Dis. Child. *60*:652-659 (Sept.) 1940.
16. Scott, E. P.: Cyanosis Caused by Methemoglobinemia, Am. J. Dis. Child. *78*:77-79 (July) 1949.
17. Stafford, G. E.: Methemoglobinemia in Infants from Water Containing High Concentration of Nitrates, Nebraska M. J. *32*:392-394 (Oct.) 1947.
18. van den Bergh, A. A. H.: Enterogene Cyanose, Deutsches Arch. f. klin. Med. *83*:86-106 (May 18) 1905.
19. Weart, J. G.: Effect of Nitrates in Rural Water Supplies on Infant Health, Illinois M. J. *93*:131-133 (March) 1948.
20. Woolley, P. V., Jr.: Methemoglobinemia Due to Well Water, Conferences at Children's Hospital of Michigan, J. Pediat. *33*:506-507 (Oct.) 1948.
21. Zo Bell, C. E.: Factors Influencing Reduction of Nitrates and Nitrites by Bacteria in Semisolid Media, J. Bact. *24*:273-281 (Oct.), 1932.

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ABSTRACT OF DISCUSSION

DR. T. ROWLAND BARNETT, Hillsboro: Another case of well-water methemoglobinemia can be added to those cases reported by Dr. Carlisle.

A baby girl was born February 23, 1950, by normal delivery, weighing 6 pounds 2.5 ounces. When she left the hospital, she was being breast fed but had to have some supplementary feedings. March 19 she entered the hospital because of noticeable cyanosis, which disappeared after twelve hours under oxygen. The baby was having some feeding trouble with the breasts and had not been gaining weight. She was placed on a formula of evaporated milk, water, and Dextrimaltose. At this time she had no fever or evidence of respiratory infection.

March 30 the baby was again brought to the hospital with cyanosis which increased on crying, with no fever, and with a heart beat of 170 per minute. The baby was again placed on oxygen, and it required forty-eight hours before the cyanosis cleared. The red blood cell count was 3,650,000 per cubic millimeter, the white blood cell count 21,550, with 67 per cent polymorphonuclear leukocytes and 33 per cent lymphocytes. Insufficient blood was obtained to determine the methemoglobin level. Roentgen-ray examination showed moderate enlargement of the thymus, and roentgen therapy was instituted.

When the baby was dismissed, she was kept on the same formula but prepared only from the city water which she had had in the hospital. To date she has had no other attack.

The well water which was being used originally in the baby's formula came from a well 20 feet deep with a nitrate content of 750 parts per million and a nitrite content of 35 parts per million. The well was 1 mile across a pasture from the well concerned in one of Dr. Carlisle's cases.

Preventive for Migraine Developed

A mixture of carbon dioxide and oxygen inhaled by the patient may interrupt migraine attacks before headache develops, a study made by two New York doctors indicates.

In the January issue of *Archives of Neurology and Psychiatry* Drs. Robert M. Marcussen and Harold G. Wolff report on a total of twenty-five trials carried out on 15 patients. Carbon dioxide in 10 per cent concentration was administered by face mask to recumbent patients for three periods of five minutes each.

"In five instances only vasoconstrictor phenomena were present. In these, carbon dioxide-air mixtures produced transient clearing of the visual disturbances with return of symptoms to their former intensity within five minutes after inhalation of the gas was discontinued.

"When the carbon dioxide-air combination was followed by the carbon dioxide-oxygen mixture in similar amounts or when the carbon dioxide-oxygen mixture was used alone, vision cleared completely and remained so. The expected headache did not follow."

The doctors report that one subject experiences extreme sleepiness and nausea as preheadache manifestations. During inhalation of the carbon dioxide mixture the patient became alert; her nausea disappeared in less than the usual interval; and the expected headache did not follow.

"When headache was present, the results were unpredictable. Although these experiments have not shown that the use of carbon dioxide-oxygen mixtures is entirely predictable, they indicate that the migraine attack can be interrupted before the headache develops."

USE OF ADRENAL CORTICAL HORMONE IN INFLAMMATORY EYE DISEASES

Case Report

OTTO LIPPMANN, M.D., Austin, Texas

THE recent release of adrenal cortical hormone (11-dehydro-17-hydroxycorticosterone in the acetate form) for use in hospitalized patients has opened up opportunities for widespread observations.

The following case report is submitted for three reasons:

1. To show the wide scope of usefulness of this drug in ocular diseases to the nonophthalmologist.

2. To augment the general knowledge of the endocrinologist concerning the pathophysiology of this hormone. The eye offers an unexcelled opportunity for minute microscopic observation *in vivo* of vascular, cellular, and exudative phenomena such as is found nowhere else in the body. The results of introduction of this hormone into human subjects can be followed directly by repeated slit lamp microscopy, which would be comparable to frequent biopsies—if this would be feasible—of involved tissue elsewhere.

3. To gather as many case reports as possible in this initial stage of the ophthalmologist's knowledge and to report a type of case not covered by previous publications.

PREVIOUS STUDIES

The original observations by Hench, Kendall, and their co-workers on pituitary adrenocorticotropic hormone (ACTH) and adrenal cortical hormones in rheumatoid arthritis have stimulated research on their use in a variety of other diseases of collagenous and mesenchymal tissues. Only three reports describing 20 cases have been published concerning these drugs in ophthalmologic conditions. Treatment in ocular diseases is particularly gratifying and safe because only a relatively small dosage over a comparatively short time is required. The administration of adrenal cortical hormone (cortisone) appears preferable to ACTH because of the manifold actions of the latter pituitary hormone which render its use either undesirable or dangerous in certain concurrent diseases, for example hypertension and certain renal diseases.

The hormonal treatment of certain ocular inflammations has revolutionized the weapons against those treatment-resistant conditions. No other treatment at the ophthalmologist's command can boast such regular results as relief of pain and photophobia in iritis within one hour, demonstrable start of absorption of fibrinous exudate in the anterior chamber of the eye within one hour, and improvement of visual acuity within a few hours.

Concepts of artificial fever therapy so frequently used in these ocular conditions will probably change. The fever probably acts by "acceleration of the mechanisms normally evoked in many acute stress states," according to Steffensen and his co-workers. There is evidence that this response to stress is achieved via the adrenal gland by the increased flow of adrenal 11-oxycorticosteroids. The substitution of fever therapy by hormone administration not only eliminates very unpleasant side-effects or dangerous complications in concurrent cardiac and other diseases but even produces a profound sense of well-being approaching euphoria in most hormonally treated patients. One report by Steffensen and his co-workers has successfully proved that even topical application of cortisone is effective, which may open new fields for other topical uses.

The total dosage of cortisone in a previous publication varied between 425 and 2,450 mg. The usual dosage was 300 mg. by intramuscular injection in divided doses; this was reduced to 150 mg. after improvement had started. While a few complications have occurred during treatment with ACTH, no adverse side-effects have been reported during treatment with cortisone. However, careful physical examination, observation, and laboratory studies including blood chemistry appear mandatory.

Enthusiasm for cortisone is dampened by the fact that frequent relapses of ocular inflammations have occurred after cessation of hormonal treatment. However, prompt and complete improvement could again be obtained by repeated hormone administration in smaller doses than the first time. It was postulated that these hormones may "condition" the patient to better response to future hormone medication or to older conventional methods of treatment if recurrence of the disease takes place.

The literature on hormonal treatment includes reports on the following ocular conditions: keratitis caused by congenital syphilis, keratitis with secondary uveitis, keratitis bullosa, severe keratitis after cataract extraction, chronic iridocyclitis, acute and plastic iritides, granulomatous iritis due to Boeck's sarcoid, acute choroiditis, retinitis centralis with central scotoma, and pigmentary retinosis.

CASE REPORT

W. B. S., a 57 year old white automobile mechanic, was admitted to Brackenridge Hospital on July 8, 1950.

Past History.—The patient's past history was unessential except for his eye trouble; he had had an intracapsular cataract extraction February 15, 1950, in the same hospital. The operation and postoperative course had been uneventful with the exception of a small prolapse of the temporal pillar of the iris, which occurred on the first postoperative day because of the patient's mental disorientation and consequent excessive movements. Healing was satisfactory with the exception of mild regional injection in an area of from 3 to 4 mm. diameter surrounding the iris prolapse. The iris prolapse was treated by three electrocoagulations March 1, April 25, and May 2. The patient had returned to work with good visual acuity in the eye operated upon. His vision in the right eye with the correction of +10.50 sphere, —1.25 cylinder, axis 170 was 20/25; with an addition of +2.50 sphere, he could read Jaeger's test type 2.

On June 3 the patient reported with iritis in the left (not operated) eye of four days' duration. Four days later on June 7 mild signs of iritis were found in the right (operated) eye. A protracted course of mild iritis in both eyes, which was treated with usual conservative ambulatory methods, followed. During this period the visual acuity of the right (operated) eye decreased, but the inflammatory signs were always worse in the left eye; the characteristics of the iritis remained the same.

Ocular examination on admission revealed that the adnexa, movements, and visual fields were normal in both eyes. The right eye showed mild ciliary injection, and a very small, flat piece of iris tissue was incarcerated in an otherwise well healed incision wound. There were mild keratic precipitates and visible flare and some cells in the aqueous humor. An operative iridectomy was apparent; the iris was under the influence of mydriatics. No granulomatous nodules in the iris could be seen. There was an aphakia with a mild bulging of the upper part of the vitreous humor into the anterior chamber and some diffuse vitreous haze. Fine fundus details could not be seen, but no gross inflammatory focus was present.

The left eye showed similar but more intensive inflammatory changes. The pupil was well dilated by mydriatics with the exception of one posterior synechia at the 6 o'clock position. There was an advanced senile cataract with nuclear as well as cortical opacities. Not more than a fundus reflex could be seen. The degree of inflammatory signs and visual acuity will be described later.

The admission diagnosis was bilateral chronic uveitis resembling the granulomatous type. Sympathetic ophthalmia had to be considered seriously because of previous intraocular surgery, exposure and repeated electrosurgical treatment of uveal tissue, occurrence of uveitis within a fairly short time after the procedures, and the type and course of the bilateral uveitis. The following features were against the diagnosis of sympathetic ophthalmia: uneventful postoperative healing of the right eye without any evidence of protracted iritis, a long interval of complete absence of any inflammatory signs in the eye operated upon before the onset of iritis in the other eye, and the appearance of uveitis at first in the left (not operated) eye and only a few days later in the right (operated) eye. Uveal pigment for a skin test was not available. Until the writing of this report, the etiology of the iritis had not definitely been determined.

Laboratory Data.—The following other observations were important: The tuberculin skin test (Mantoux, first strength) was positive. A Kline (exclusion) test of the blood was negative. Agglutination for undulant fever was negative. A roentgenogram of the chest was negative except for a small calcified lymph node in the right supraclavicular area. In a consultation Dr. George Clark, an internist, observed no significant abnormalities.

Treatment.—Between July 8 and 17 treatment included bed rest, atropine instillations, hot applications, oral multivitamin and aspirin medications, and intravenous injections of typhoid H antigen as fever therapy. All treatments did not influence the course of the iritis in either eye. On July 17, the patient showed mild ciliary injection of both eyes, worse in the left than right. There were fairly heavy keratic precipitates, which were worse and more numerous in the left eye than in the right, and a few cells in the aqueous fluid. There was an aqueous flare in the right eye of 1 plus and in the left eye of 2 plus. The visual acuity in the right eye with the correction of +9.75 sphere —0.75 cylinder, axis 180 was 20/100, in the left eye with the correction of —5.00 sphere —1.00 cylinder, axis 90, it was 20/400.

Intramuscular injections of "Cortone"* (11-dehydro-17-hydroxycorticosterone-21-acetate) were started July 18. The patient received 100 mg. three times daily on the first day; on the second day, 100 mg. twice daily; and on the third day, three injections of 40, 30, and 30 mg. During this period other treatments were discontinued.

After one day's treatment with Cortone on July 19, the pain in the patient's eyes had disappeared. He said that he felt as good as "a 25 year old man." The injection had decreased. The visual acuity increased to 20/70 +2 —2 in the right eye and 20/400 +1 in the left eye. There was no essential change in the aqueous flare or the cellular contents.

After two days' treatment with Cortone, hardly any injection was noted. The patient felt unusually well. The aqueous beam with the slitlamp decreased to minus in the right eye and 1 plus in the left. No cells could be found in the right aqueous humor and only a few in the aqueous fluid of the left eye. On July 21 after three days' treatment there were no cells and the visual acuity of the right eye was 20/70 +2. In spite of cessation of treatment, the improvement continued. On July 22, there was only a questionable aqueous flare in the left eye and a visual acuity of the right eye of 20/60 —1.

The patient was dismissed from the hospital July 24. There was no aqueous flare and the old keratic precipitates showed signs of disintegration. On July 26, the patient had done a little light work. Vision of the right eye was 20/50 —2 +2. The residual visual impairment was the result of vitreous opacities in the right eye and of cataract in the left eye. These kinds of opacities could not be expected to yield to hormonal treatment.

The patient's sense of well-being and the condition of his eyes had remained the same on July 28 and until the time at which this report was submitted for publication.

SUMMARY

Treatment of inflammatory conditions of the eye by adrenal cortical hormones is briefly discussed. A case of bilateral chronic uveitis, three and one-half months after cataract extraction, resembling a granulomatous type of uveitis and suggesting sympathetic ophthalmia, is reported, and its successful treatment with Cortone is described.

*Cortone is manufactured by Merck and Company.

REFERENCES

1. Albright, F.: Cushing's Syndrome, Its Pathological Physiology, Its Relationship to Adrenogenital Syndrome, and Its Connection with

CORTISONE IN EYE DISEASE — Lippmann — *continued*

Problem of Reaction of Body to Injurious Agents ("Alarm Reaction" of Selye), Harvey Lectures, 1942-1943, Baltimore, Williams & Wilkins, 1943.

2. Gordon, D. M., and McLean, J. M.: Effects of Pituitary Adrenocorticotropic Hormone (ACTH) Therapy in Ophthalmologic Conditions, J.A.M.A. *142:*1271-1276 (April 22) 1950.

3. Hench, P. S.; Kendall, E. C.; Slocumb, C. H.; and Polley, H. F.: Effect of Hormone of Adrenal Cortex (17-Hydroxy-11-Dehydrocorticosterone: Compound E) and of Pituitary Adrenocorticotropic Hormone on Rheumatoid Arthritis, Proc. Staff Meet., Mayo Clin. *24:* 181-197 (April 13) 1949.

4. Kendall, E. C.: Some Observations on Hormone of Adrenal Cortex Designated Compound E, Proc. Staff Meet., Mayo Clin. *24:* 298-301 (May 25) 1949.

5. Olson, J. A., and others: Effect of ACTH on Certain Inflammatory Diseases of Eye, J.A.M.A. *142:*1276-1278 (April 22) 1950.

6. Selye, H.: General Adaptation Syndrome and Diseases of Syndrome, J. Clin. Endocrinol. *6:*117-230 (Feb.) 1946.

7. Steffensen, E. M., and others: Experimental Use of Cortisone in Inflammatory Eye Disease, Am. J. Ophth. *33:*1033-1040 (July) 1950.

8. Vail, D.: American Ophthalmological Society Meeting, editorial, Am. J. Ophth. *33:*1158-1160 (July) 1950.

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ACTH AND COLCHICINE IN THERAPY OF GOUT

Report of a Case of Acute Gouty Arthritis

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THERE is little excuse to repeat the time-honored descriptions and aphorisms which have been accumulated on gout. For an accurate description of this disease and its clinical manifestations one may refer to the classic paper of Thomas Sydenham (1624-1689).[8]

Philip S. Hench, a pioneer in the field of gout, has summarized in published form[5, 6] his many years of exhaustive study, and John H. Talbott has presented a complete discussion of gout, which was published by Oxford Medical Publications,[10] and later a follow-up study, which appeared in *Postgraduate Medicine*.[11] Therefore, it would serve no useful purpose to reiterate here those facts which are well known and readily available.

A survey taken recently of some of my patients with gout demonstrated that, even though most of them have had this condition for a number of years, few could manage the disease. The majority were not certain that diet was of any value in the treatment of the disease, and none was satisfied with the present status of its care. Many paradoxes were encountered; for example, in my experience this disease has always been associated with importance and prosperity, but Talbott's series was obtained from the indigent and unemployed in Boston. The fact of heredity has always been emphasized, accepted, and yet perhaps not completely proved. Although gout has been predominantly a disease of males, particularly those in their third and fourth decades, it does occur in both sexes in people as young as 7 years and as old as 80.

COLCHICINE

A number of years ago Hench proposed the alternate use of one of the cinchophen products and large doses of salicylate in the treatment of gout, and a short time later he found that the output of urates was increased when glycocoll was added to the treatment.[5] The results in the treatment of chronic gouty arthritis were better by this method than by any previous treatment, but a little later Talbott emphasized the use of colchicine in the acute attack and also urged that it be used in the control of chronic gouty arthritis.[9] Among others, Elmer C. Bartels advanced the theory that a diet low not only in purines but also in fats was necessary for the control of gouty arthritis and that this treatment was more effective when supplemented by the use of cinchophen.[1] Talbott was reluctant to abandon the use of colchicine, and the Arthritis Institute of the University of Michigan, at that time under the leadership of Richard Freyberg, investigated the properties of this drug.[3]

Colchicum was known to the Byzantine physicians (fifth, sixth, and seventh centuries) under the name of hermodactyl (finger of Hermes). Jacobus Psychrestus, a physician of Constantinople, employed it for painful articular attacks. Physicians of the medical school of Salerno used it for arthritis in the twelfth century and knew that it helped arthritic gout and pellagra. Baron Anton von Storck in 1763 introduced the use of Colchicum autumnale (named from the ancient Greeks because the plant grew in Colchis, Asia Minor[2]) or meadow saffron, and showed that it could be given with impunity. Quacks quickly flooded the market with gout specifics, some of which are still used as Liqueur Anti-Goutt eux du Docteur Laville, Reynold's Specific, Albert's Remedy, and so forth. Benjamin Franklin is credited with introducing colchicum into this country.

Colchicine (the alkaloid of colchicum) was isolated by Pelletier and Caventou in 1820. Its mode of action is still not understood. It is not a diuretic; it has no effect on the kidneys; it has no influence on the metabolism of purine bodies; it does not influence the excretion of urates or decrease their blood level. It is not an analgesic because it fails to relieve pain of nongouty origin. It should be given with caution to the elderly and those with certain diseases of the kidney, heart, liver, or gastrointestinal tract. It may be used to abort a gouty attack.

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ACTH

In 1946, researchers with Armour and Company in Chicago isolated a substance which they called adrenocorticotropin; they sent this material to George W. Thorn, who found that it produced certain physiologic and metabolic effects.[12] It was next used by Fuller Allbright and his associates, and it was found to be of particular interest in the study of two diseases of altered metabolism, namely, myasthenia gravis and gout. Outstanding in the study of this hormone in uric acid metabolism and gout has been the work of William Q. Wolfson, L. Hellman, Jerome Conn of the University of Michigan, J. H. Talbott now of the University of Buffalo, Walter Bauer, and Marion Ropes of Harvard. The product became known as ACTH, or the adrenocorticotropic hormone, with the realization that there may be more than one of these hormones. These are elaborated by the pituitary gland under the influence and regulation of the hypothalamus, and it is stated that this center is to be found in certain cells of the paraventricular nuclei. Under this stimulus certain areas of the adrenal cortex produce 11-oxysteroids which are similar to cortisone or compound E.

Investigators have discovered that there are several differences in the hormonal behavior of gouty and nongouty persons. The gouty person produces an abnormal androgen and also eventually has a decreased 17-ketosteroid production. It was found that under conditions of stress the gouty person failed to produce a normal rebound response of 11-oxysteroids after the administration of ACTH.[12]

It was discovered that an attack of acute gouty arthritis could be precipitated in the gouty person by the administration and withdrawal of ACTH[4] and also that these attacks could be stopped by the administration of an additional dose of ACTH. It was observed that an attack of acute gouty arthritis could be terminated by 50 mg. of ACTH in approximately 70 per cent of patients. Others required a second dose; a few required a third dose; but almost all could be controlled by a fourth dose. The simultaneous administration of colchicine and ACTH followed by the continued administration of the maximum amount of colchicine tolerated would prevent the recurrence of gout. It was also discovered that the continued administration of ACTH resulted in at least partial removal of tophaceous deposits.

Four hours after the administration of ACTH patients with acute gouty arthritis could feel relief by the minute rather than by the day. The characteristic depression and alteration in mood that accompanies the appearance of acute gouty arthritis rapidly would give way to the appearance of the typical good-natured, extrovert behavior of the gouty person.

Because of the dramatic relief obtained with ACTH in the treatment of acute gouty arthritis, it was decided that, even though the supply of this hormone was extremely limited, as much would be released for the treatment of gout as was possible.[12]

TREATMENT

At the onset of the attack, 50 mg. of ACTH is administered subcutaneously and .00065 Gm. of colchicine is given after each meal and at bedtime until the first loose stool occurs. For those cases not responding within four hours, a second 50 mg. dose of ACTH is given; and those rare patients not responding to this treatment are given a third or fourth dose. Colchicine is decreased to the maximum amount tolerated by the patient for an indefinite period of time, which may be two to four tablets daily. If the patient is intolerant to oral colchicine, the intravenous product made by Lilly may be used, the dosage usually being .5 mg. intravenously. Wolfson prefers the products made by Lilly and states that they seem to be more equal in potency. He has termed the above procedure "colchicinization."

No special diet is prescribed, but alcohol is eliminated because it is oxidized to fat. Gradual reduction to a normal weight is desirable, but rapid reduction is undesirable. The usual measures and warnings about exercise, care of the feet, chilling, and emotional disturbances are exercised. Tophaceous deposits may be removed only after complete colchicinization has been accomplished.

ACTH relieves the attack of gout but does not cause normal urate metabolism. Urates are decreased and oxypurines are considerably increased. It has been impossible to obtain normal urate and oxypurine levels in a gouty person through the use of ACTH. Lack of 11-oxysteroid seems to precipitate acute gouty arthritis whether spontaneously occurring or after ACTH withdrawal in interval gout.

It is stated that an unexplained attack of gout often means coronary occlusion. Coronary occlusion often terminates the life of the gouty person as does nephritis.

CASE REPORT

A 40 year old white woman was seen April 15, 1950, complaining of severe pain in the metatarsal joints of both feet for fifteen hours. The temperature and blood pressure were normal; results of a physical examination were essentially normal except that there was slight redness and swelling of the metatarsal joints of both feet, redness and swelling of the right metatarsophalangeal joint, and considerable warmth of the same joints. Uric acid was 4.5 mg. per 100 cc. of blood. A tophus was removed from the right index finger and several leading pathologists of this area concurred in the opinion that it constituted a true tophus.

Past History.—The patient had suffered from arthritis for approximately twenty years. She stated that she had previously been treated for hypertrophic arthritis and question-

able rheumatoid arthritis with salicylates, gold therapy, physiotherapy, sulfonamide therapy, and various vaccines.

Roentgen-Ray Data.—Roentgenograms showed evidence of mixed arthritis (hypertrophic and atrophic arthritis), but the radiologist stated there seemed to be nothing diagnostic of gouty arthritis.

Treatment.—The patient was given 50 mg. of ACTH intramuscularly and colchicine was started, one tablet of .00065 Gm. (Lilly) four times a day. Marked relief of pain occurred within four hours, but it required three injections (50 mg. of each) of ACTH before maximum benefit was obtained. Approximately 90 per cent relief of pain occurred within twelve hours, but the greatest percentage of relief occurred within the first four hours. Colchicine was continued until the patient developed diarrhea, and then the dose was reduced to three tablets of colchicine daily. Further diarrhea occurred on this dosage and her maintenance dose was found to be two tablets daily, which she has continued taking since the acute attack of gouty arthritis.

Results.—Within four hours after the first injection of ACTH the patient had definite relief and could walk without too much discomfort. She has not had a severe attack of gouty arthritis since the administration of ACTH and colchicinization. She has had slight pain, tenderness, swelling, and redness of the metatarsal joints of both feet since the acute attack, but the pain has not been severe enough to cause her to go to bed. She feels that colchicinization has prevented further attacks of acute gouty arthritis.

The small amount of residual discomfort this patient has may be accounted for on the basis of preexisting osteoarthritis.

Laboratory Data (Post-Treatment).—Approximately one week after the acute attack of gouty arthritis subsided, additional laboratory work was done. The blood count, sedimentation rate, serum protein with albumin-globulin ratio, fasting blood sugar levels, and hematocrit level were normal. Blood uric acid, however, was 0.6 mg. per 100 cc. as compared to 4.5 mg. before treatment. A twenty-four hour urine specimen showed the following: urate excretion 0.22 Gm., creatinine excretion 6 mg., no glycosuria, and 17-ketosteroid 4.6 mg.

SUMMARY

The use of colchicine and ACTH in the treatment of gout is briefly reviewed. A case of acute gouty arthritis successfully treated with those drugs is reported.

REFERENCES

1. Bartels, E. C.: Successful Treatment of Gout, Ann. Int. Med. *18*:21-28 (Jan.) 1943.
2. Comroe, B. I.: Gout, in Arthritis and Allied Conditions, ed. 3, Philadelphia, Lea and Febiger, 1944.
3. Freyberg, R.: Personal communication, March, 1950.
4. Hellman, L.: Production of Acute Gouty Arthritis by Adrenocorticotropin, Science *109*:280-281 (March 18) 1949.
5. Hench, P. S.: Diagnosis and Treatment of Gout and Gouty Arthritis, J.A.M.A. *116*:453-459 (Feb. 8) 1941.
6. Hench, P. S.: Gout and Gouty Arthritis, in R. L. Cecil: Textbook of Medicine, ed. 7, Philadelphia, W. B. Saunders, 1947, pp. 673-685.
7. Mote, J. R.: Proceedings of First Clinical ACTH Conference, Philadelphia and Toronto, Blakiston and Company, 1950.
8. Sydenham, T.: Works of Thomas Sydenham, M. D., translated from the Latin edition by Dr. Greenhill by R. S. Latham, London, C. and J. Adlard, Printers, n. d.
9. Talbott, J. H.: Treatment of Gout with Special Reference to Colchicine, Bull. New York Acad. Med. *18*:318-328 (May) 1942.
10. Talbott, J. H.: Gout, edited by H. A. Christian, New York, Oxford University Press, 1943.
11. Talbott, J. H.: Diagnosis and Treatment of Gouty Arthritis, Postgrad. Med. *5*:386-393 (May) 1949.
12. Wolfson, W. Q., and Cohn, C.: Role of Pituitary Adrenocorticotropic Hormone (ACTH) and of Adrenal Cortical Steroid Hormones in Pathological Physiology and Experimental Therapeutics of Clinical Gout, in Mote.[7]

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EXFOLIATIVE DERMATITIS FROM DIHYDROSTREPTOMYCIN

Report of Case

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MANY types of cutaneous lesions have been described as occurring in the course of streptomycin therapy.[1, 3, 5, 8, 9, 10, 11] These include instances of the contact allergic type of eczematous dermatitis, atopy, urticaria and the id type of reaction. However, only two authentic instances of exfoliative dermatitis have been reported; one by Harris and Walley[4] and the other by McCaffery and Hess[6], the latter resulting in a fatal outcome.

Following is a case report of exfoliative dermatitis as the result of streptomycin. The possibility that streptomycin or dihydrostreptomycin may contain an antigen closely related to penicillin is suggested by this case.

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CASE REPORT

A 56 year old white man was admitted to the Veterans Administration Hospital, Aspinwall, Pa., on September 7, 1949. He was acutely ill with his chief complaint a rash over the entire body.

Past History.—The patient had been discharged from the hospital August 20 after a twenty day course of dihydrostreptomycin,† 1 Gm. daily, for a nodular lesion on the liver

†*Dihydrostreptomycin Hydrochloride (Squibb).*

which was thought to be tuberculous. During that period beginning May 30, 1949, he had received 4,500,000 units of penicillin for syphilis, since the Kahn blood test was positive. On August 22, two days after his discharge from the hospital, he noted a slight generalized itching of the skin. A few days later an erythema of the trunk and extremities appeared.

Physical Examination.—On the second admission September 7, a generalized erythematous maculopapular dermatitis accompanied by marked itching and burning was present. Blebs and vesicles of varying size appeared over the trunk, buttocks, and extremities. These ruptured, leaving large, red, raw looking areas. The entire body was covered with a diffuse erythematous, vesicular eruption, most pronounced over the trunk and extremities, presenting a violaceous hue. The face was swollen and red and there was edema of the eyelids and extremities (fig. 1a). The mucous membrane of the mouth showed erosive areas.

Laboratory examinations were as follows: red blood cell count 3,800,000 per cubic millimeter, hemoglobin 11 Gm. per 100 cc. of blood, white blood cells count 18,000 per cubic millimeter with a differential of 51 per cent neutrophils, 25 per cent lymphocytes, 6 per cent monocytes, 7 per cent eosinophils, and 1 per cent basophils. The Kahn and Kolmer tests were positive. The urinalysis showed a trace of albumin.

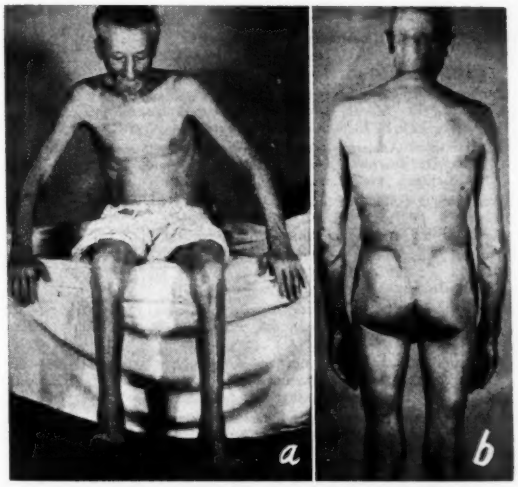

FIG. 1a. A photograph of the patient on admission September 7, 1949, showing exfoliation, edema, and a normal amount of hair.
b. A photograph taken five weeks later showing the exfoliation completed, with baldness and complete loss of body hair and nails.

The clinical course was stormy and the patient continued to be seriously ill. His temperature continued to be elevated, reaching 104.4 F. during the first part of October. A roentgenogram showed widespread bronchopneumonia with bilateral pleural effusion. At this time the white blood count was 23,500 per cubic millimeter with a differential of 66 per cent neutrophils, 10 per cent lymphocytes, 2 per cent monocytes, and 22 per cent eosinophils. The patient received aureomycin 0.25 Gm. every six hours. The skin manifestations continued and the exfoliation became extensive and complete, including loss of all hair and nails. The pneumonic process, however, responded to aureomycin and gradually the dermatitis receded after five weeks (fig 1b).

Intracutaneous and contact tests were done after the skin manifestations had cleared, including patch and direct intracutaneous skin tests as well as passive transfer tests with penicillin, streptomycin, dihydrostreptomycin, Trichophytin and Oidiomycin. Delayed reaction tuberculin tests were also performed. The results were as follows: The direct and indirect tests were uniformly negative. There was a positive delayed skin reaction of the tuberculin type to streptomycin and dihydrostreptomycin and a positive patch test to the same substances. In addition scrapings and cultures of the skin of the feet were examined for fungi and no pathogens were found. The collodion particle technique of Cavelti[2] for specific antibodies to streptomycin was negative.

DISCUSSION

Allergic reactions to the antibiotic drugs have been divided into the serum disease pattern, the anaphylactic type of reaction, atopic sensitivity, the id type of reaction, and the contact allergic type of eczematous dermatitis. This case demonstrates a delayed type of reaction, namely of the id variety, morphologically resembling exfoliative dermatitis. The patient had received a course of penicillin prior to administration of the dihydrostreptomycin. Peck and Siegal[7] pointed out that there is a cross sensitization between penicillin and streptomycin; hence, it is likely that sensitization was produced by the penicillin so that subsequent treatment with streptomycin produced the delayed id type of reaction.

SUMMARY

A case of severe exfoliative dermatitis in a 56 year old white man after parenteral therapy with dihydrostreptomycin which had been preceded by penicillin treatment is reported. A tuberculin type of cutaneous reaction resulted and the relationship of this reaction is discussed. Severe exfoliative dermatitis as the result of the treatment with streptomycin and penicillin is described.

REFERENCES

1. Besancon, J. L.; Klotz, H. P.; and Debray, J.: Sensitization Disorders Caused by Streptomycin in Nursing Personnel, Semaine d. hôp. Paris *25*:2389 (July 30) 1949.
2. Cavelti, P. A.: Technic of Collodion Particle Agglutination, J. Immunol. *57*:141-154 (Oct.) 1947.
3. Criep, L. H.: Streptomycin and Extrapulmonary Tuberculosis; Reactions to Penicillin and Streptomycin, Nat. Tuberc. A. Tr., 1949, pp. 1-7.
4. Harris, W. C., and Walley, R. V.: Exfoliative Dermatitis Complicating Streptomycin Therapy, Lancet *1*:112-114 (Jan.) 1950.
5. Hettig, R. A., and Adcock, J. D.: Studies on Toxicity of Streptomycin for Man; Preliminary Report, Science *103*:335-357 (March 22) 1946.
6. McCaffery, R. J., and Hess, E.: Fatal Exfoliative Dermatitis Due to Treatment of Urinary Tuberculosis with Streptomycin and Moogrol, Urol. & Cutan. Rev. *52*:525-528 (Sept.) 1948.
7. Peck, S. M., and Siegal, S.: Immunologic Relationships of Antibiotics and Trichophytin; Clinical Observations and Animal Experiments, J. Invest. Dermat. *9*:165-185 (Oct.) 1947.
8. Simon, S. W.: Skin Sensitivity to Streptomycin, J. Allergy *20*: 56-61 (Jan.) 1949.
9. Smith, G. C.: Epidermal Sensitivity Due to Streptomycin, J. South Carolina M. A. *44*:164-165 (May) 1948.
10. Strauss, M. J., and Warring, F. C., Jr.: Epidermal Sensitization to Streptomycin: Report of 6 Cases Occurring in 12 Nurses Handling the Drug. J. Invest. Dermat. *9*:99-106 (Aug.) 1947.
11. Streptomycin, Report of Council on Pharmacy and Chemistry, J.A.M.A. *133*:320-321 (Feb. 11) 1947.

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NEWS

COMING MEETINGS AND CLINICS

State Medical Association of Texas, Galveston, May 1-2, 1951. Dr. William M. Gambrell, Austin, Pres.; Tod Bates, 700 Guadalupe St., Austin, Executive Secy.

American Medical Association, Cleveland, Dec. 5-8, 1950. Dr. Elmer L. Henderson, Louisville, Ky., Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy, New York, Feb. 5-7, 1951. Dr. Theodore L. Squier, Milwaukee, Pres.; Mr. James O. Kelley, 208 E. Wisconsin Ave., Milwaukee 2, Executive Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 9-14, 1950. Dr. Earl D. Osborne, Buffalo, N. Y., Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave. N. W., Cleveland 15, Secy.

American Academy of General Practice, San Francisco, March 19-22, 1951. Stanley R. Truman, Oakland, Calif., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Sec'y.

American Academy of Neurological Surgery. Dr. William S. Keith, Toronto, Canada, Pres.; Dr. T. C. Erickson, 1300 University Ave., Madison 5, Wis., Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 8-13, 1950. Dr. Conrad Berens, New York, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Chicago, Oct. 16-19, 1950. Dr. Edward B. Shaw, San Francisco, Pres.; Dr. C. G. Grulee, 636 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951. Dr. Alfred Blalock, Baltimore, Pres.; Dr. Brian Blades, 901 23rd St. N. W., Washington, D. C., Secy.

American Association of Genito-Urinary Surgeons, Skytop, Pa., May 16-18, 1951. Dr. Roger C. Graves, Boston, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, Hot Springs, Va., Sept. 7-9, 1950. Dr. S. A. Cosgrove, Jersey City, N. J., Pres.; Dr. L. A. Calkins, University of Kansas Medical Center, Kansas City 3, Secy.

American Cancer Society, New York, Oct. 27, 1950. Dr. Alton Ochsner, New Orleans, Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Exec. Vice-Pres.

American College of Chest Physicians, Atlantic City, N. J., June 7-10, 1951. Dr. Louis Mark, Columbus, Ohio, Pres.; Mr. Murray Kornfeld, 500 N. Dearborn St., Chicago 10, Executive Secy.

American College of Physicians, St. Louis, April 9-13, 1951. Dr. William S. Middleton, Madison, Wis., Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology. Dr. C. Edgar Virden, Kansas City, Mo., Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons, Boston, Oct. 23-27, 1950. Dr. Frederick A. Coller, Ann Arbor, Mich., Pres.; Dr. Paul B. Magnuson, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine, Boston, Aug. 28-Sept. 1, 1950. Dr. Earl C. Elkins, Rochester, Minn., Pres.; Dr. Richard Kovacs, 2 E. 88th St., New York 28, Secy.

American Dermatological Association, Hot Springs, Va., May 23-26, 1951. Dr. Henry E. Michelson, Minneapolis, Pres.; Dr. L. A. Brunsting, 102 2nd Ave., S. W., Rochester, Minn, Secy.

American Gastro-Enterological Association, Atlantic City, June 8-9, 1951. Dr. John G. Mateer, Detroit, Pres.; Dr. Dwight L. Wilbur, 655 Sutter St., San Francisco, Secy.

American Gynecological Society, New York, May 7-9, 1951. Dr. Frederick Irving, Brookline, Pres.; Dr. Norman F. Miller, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Hospital Association, Atlantic City, N. J., Sept. 18-21, 1950. Mr. John Hatfield, Philadelphia, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, White Sulphur Springs, W. Va., May 6-8, 1951. Dr. Louis H. Clerf, Philadelphia, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 8, N. Y., Secy.

American Neurological Association. Dr. Wilder G. Penfield, Montreal, Canada, Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, White Sulphur Springs, W. Va., June 7-9, 1951. Dr. John H. Dunnington, New York, Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, White Sulphur Springs, W. Va., June 20-23, 1951. Dr. James S. Speed, Memphis, Pres.; Dr. C. Leslie Mitchell, Henry Ford Hospital, Detroit 2, Secy.

American Pediatric Society, Atlantic City, May 2-5, 1951. Dr. Bronson Crothers, Boston, Pres.; Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12, Secy.

American Proctologic Society. Dr. Hoyt R. Allen, Little Rock, Pres.; Dr. W. Wendell Green, 1838 Parkwood Ave., Toledo 2, Secy.

American Psychiatric Association. Dr. John C. Whitehorn, Baltimore, Pres.; Dr. R. Finley Gayle, 501 E. Franklin St., Richmond, Va., Secy.

American Public Health Association, St. Louis, Oct. 30-Nov. 3, 1950. Dr. Lowell J. Reed, Baltimore, Pres.; Dr. R. M. Atwater, 1790 Broadway, New York 19, Secy.

American Society of Anesthesiologists, Houston, Nov. 7-10, 1950. Dr. Rolland J. Whitacre, East Cleveland, Ohio, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists, Chicago, Oct. 17-21, 1950. Dr. James B. McNaught, Denver, Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Washington, D. C., April 11-13, 1951. Dr. Samuel C. Harvy, New Haven, Conn., Pres.; Dr. Nathan Womack, University of Iowa, Iowa City, Secy.

American Urological Association, Chicago, May 21-24, 1951. Dr. Thomas D. Moore, Memphis, Tenn., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Houston, Oct. 5-7, 1950. Dr. Joseph C. Bunten, Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter, Cleveland, Oct. 31-Nov. 3, 1950. Dr. H. W. Meyerding, Rochester, Minn., Pres.; Dr. Arnold S. Jackson, Jackson Clinic, Madison 5, Wis., Secy.

National Tuberculosis Association, Cincinnati, May 14-18, 1951. Dr. David T. Smith, Durham, N. C., Pres.; Dr. H. Stuart Willis, 1790 Broadway, New York 19, Secy.

Radiological Society of North America, Chicago, Dec. 10-15, 1950. Dr. Warren W. Furey, Chicago, Pres.; Dr. D. S. Childs, Medical Arts Bldg., Syracuse 2, N. Y., Secy.

Southern Medical Association, St. Louis, Nov. 13-16, 1950. Dr. Hamilton W. McKay, Charlotte, N. C., Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham, Ala., Secy.

Southern Psychiatric Association. Dr. R. Finley Gayle, Jr., Richmond, Va., Pres.; Dr. Newdigate M. Owensby, Medical Arts Bldg., Atlanta, Ga., Secy.

Southern Surgical Association, Hollywood, Fla., Dec. 5-7, 1950. Dr. G. V. Brindley, Temple, Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville, Tenn., Secy.

Southwest Allergy Forum, San Antonio, April 8-10, 1951. Dr. J. H. W. Rouse, San Antonio, Pres.; Dr. Boen Swinny, 224 Medical Arts Bldg., San Antonio 5, Secy.

Southwest Regional Cancer Conference, Fort Worth, Nov. 15, 1950. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association, Phoenix, Ariz., Oct. 26-28, 1950. Dr. I. J. Marshall, Roswell, N. Mex., Pres.; Dr. W. W. Schuessler, 1415 First National Bank Bldg., El Paso, Secy.

Southwestern Surgical Congress, Denver, Sept. 25-27, 1950. Dr. Thomas G. Orr, Kansas City, Pres.; Dr. C. R. Rountree, 210 Plaza Court, Oklahoma City 3, Secy.

Tri-State Medical Assembly, Shreveport, La., Sept. 29-30, 1950. Dr. Charles Gowen, Shreveport, Pres.; Dr. John Walter Jones, 401 E. Fifth St., Texarkana, Secy.

United States-Mexico Border Public Health Association, Los Angeles, April 4-6, 1951. Dr. Luis Arriaga Velez, Chihuahua, Mexico, Pres.; Dr. M. F. Haralson, 314 U. S. Court House, El Paso, Secy.

STATE

Texas Academy of General Practice, Fort Worth, Sept. 25, 1950. Dr. H. T. Jackson, Fort Worth, Pres.; Dr. W. P. Higgins, Jr., Medical Arts Bldg., Fort Worth, Secy.

Texas Academy of Internal Medicine, Houston, Dec. 9-10, 1950. Dr. Samuel A. Shelburne, Dallas, Pres.; Dr. John S. Chapman, 3810 Swiss Ave., Dallas, Secy. Meeting restricted to members.

Texas Air-Medics Association, Galveston, April 30, 1951. Dr. D. P. Laugenour, Dallas, Pres.; Dr. C. F. Miller, 906 Medical Arts Bldg., Waco, Secy.

Texas Association of Obstetricians and Gynecologists, Galveston, Feb. 1951. Dr. Howard Smith, Marlin, Pres.; Dr. George F. Adam, 4115 Fannin. Houston. Secy.

Texas Chapter, American College of Chest Physicians, Galveston, April 30, 1951. Dr. David McCullough, Kerrville, Pres.; Dr. Henry R. Hoskins, 514 Medical Arts Bldg., San Antonio, Secy.

Texas Club of Internists. Dr. Ghent Graves, Houston, Pres.; Dr. Hatch W. Cummings, Jr., Houston, Secy.

Texas Dermatological Society, Galveston, April 30, 1951. Dr. J. G. Brau, Dallas, Pres.; Dr. W. Harris Connor, 601 Medical Arts Bldg., Houston, Secy.

Texas Diabetes Association, Galveston, April 29, 1951. Dr. B. F. Smith, Houston, Pres.; Dr. W. N. Powell, W. Ave. F, Temple, Secy.

Texas Division, American Cancer Society, Galveston, Nov. 6, 1950. Mr. Frank C. Smith, Houston, Pres.; Mr. J. Louis Neff, 2307 Helena St., Houston 6, Executive Director.

Texas Heart Association, Galveston, April 30, 1951. Dr. George W. Parson, Texarkana, Pres.; Miss Roberta Miller, 411 Reserve Loan Life Bldg., Dallas, Executive Secy.

Texas Hospital Association, San Antonio, April 24-26, 1951. Mr. Roy Wilmesmeier, Houston, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association, San Antonio, Oct. 6, 1950. Dr. A. T. Hanretta, Austin, Pres.; Dr. David Wade, 510 Capital National Bank, Austin, Secy.

Texas Orthopedic Association, Galveston, April 30, 1951. Dr. Louis Breck, El Paso, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Fort Worth, Oct. 6-7, 1950. Dr. J. E. Ashby, Dallas, Pres.; Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Secy.

Texas Public Health Association, Galveston, Feb. 18-21, 1951. Mr. Barnie A. Young, Austin, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society, Galveston, Jan. 19-20, 1951. Wayne D. Ramsey, Abilene, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association, Galveston, April 30, 1951. Dr. Joe Gandy, Houston, Pres.; Dr. W. F. Parsons, First National Bank Bldg., Fort Worth, Secy.

Texas Rheumatism Association, Houston, Dec. 8, 1950. Dr. Moise D. Levy, Jr., Houston, Pres.; Dr. Robert H. Mitchell, 210 Medical Arts Bldg., Fort Worth, Secy.

Texas Society for Mental Health, Galveston, March 8-9, 1951. Dr. Hamilton Ford, Galveston, Pres.; Mrs. Elizabeth F. Gardner, 1617 Watchhill Road, Austin 21, Executive Secy.

Texas Society of Anesthesiologists. Galveston, April 30, 1951. Dr. Russell Bonham, Houston, Pres.; Dr. Werner Hoeflich, 2301 Reba, Houston, Secy.

Texas Society of Gastroenterologists and Proctologists, Galveston, April 30. 1951. Dr. Carl G. Giesecke, San Antonio, Pres.; Dr. John S. Bagwell, Medical Arts Bldg., Dallas, Secy.

Texas Society of Ophthalmology and Otolaryngology, Dallas, Dec. 8-9, 1950. Dr. V. R. Hurst, Longview, Pres.; Dr. John L. Matthews, 929 Nix Professional Bldg., San Antonio, Secy.

Texas Society of Pathologists, Galveston, Jan. 28, 1951. Dr. Charles Phillips, Temple, Pres.; Dr. A. O. Severance, 205 Camden, San Antonio, Secy.

Texas Surgical Society, Waco, Oct. 2-3, 1950. Dr. R. J. White, Fort Worth, Pres.; Dr. Truman G. Blocker, 927 Strand, Galveston, Secy.

Texas Tuberculosis Association, Dallas, April 13-14, 1951. Dr. Elliott Mendenhall, Dallas, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society, San Antonio, Jan. 22, 1951. Dr. Hub E. Isaacks, Fort Worth, Pres.; Dr. John M. Pace, 428 Medical Arts Bldg., Dallas, Secy.

DISTRICT

Second District Society. Dr. James W. Rainer, Odessa, Pres.; Dr. Frank M. James, 1021 N. Whitaker Ave., Odessa, Secy.

Third District Society, Lubbock, Oct. 3-4, 1950. Dr. Allen T. Stewart, Lubbock, Pres.; Dr. James T. Hall, 1302 Avenue Q, Lubbock, Secy.

Fourth District Society, Brownwood, Nov. 1. 1950. Dr. Gordon F. Madding, San Angelo, Pres.; Dr. S. B. Locker, First National Bank Bldg., Brownwood, Secy.

Fifth and Sixth Districts Society, Corpus Christi, 1951. Dr. E. King Gill, Corpus Christi, Pres.; Dr. Foy Moody, 1611 Fifth St., Corpus Christi, Secy.

Seventh District Society. Dr. John F. Thomas, Austin, Pres.; Dr. George W. Tipton, 502 W. 15, Austin, Secy.

Eighth District Medical Society, Spring, 1951. Dr. Leonard Johnson, El Campo, Pres.; Dr. Robert Casey, Texas City, Secy.

Tenth District Medical Society, Port Arthur. Dr. J. A. Richardson, Sr., Jasper, Pres.; Dr. Dale H. Davies, Liberty, Secy.

Eleventh District Society, Tyler, Oct. 26, 1950. Dr. E. G. Faber, Tyler, Pres.; Dr. John M. Travis, Jr., Jacksonville, Secy.

Twelfth District Society, Temple, Jan. 9, 1951. Dr. W. K. Logsdon, Corsicana, Pres.; Dr. N. C. Smith, Hillsboro, Secy.

Thirteenth District Society, Mineral Wells, Oct. 10, 1950. Dr. R. L. Daily, Wichita Falls, Pres.; Dr. S. W. Wilson, Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society, Bonham, June, 1951. Dr. Mayo Tenery, Waxahachie, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, Texarkana, Oct. 26, 1950. Dr. F. V. Mondrik, Longview, Pres.; Dr. Hardy Cook, Longview, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 26-29, 1951. Miss Betty Elmer, Medical Arts Bldg., Dallas 1, Executive Secy.

International Post-Graduate Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1951. Dr. John J. Hinchey, P. O. Box 2445, San Antonio, Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 5-8, 1951. Dr. Woodard D. Beacham, Room 105, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Sept. 20, 1950. Dr. Robert L. Daily, 214 Hamilton Bldg., Wichita Falls, Program Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 30-Nov. 2, 1950. Mrs. Muriel R. Waller, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Post Graduate Medical Assembly of South Texas, Houston, Nov. 20-22, 1950. Dr. Donald M. Paton, Secy., 229 Medical Arts Bldg., Houston.

ASSOCIATION OF AMERICAN PHYSICIANS AND SURGEONS

The annual meeting of the Association of American Physicians and Surgeons will be held October 5-7 at the Shamrock Hotel, Houston. The theme for the meeting will be "Freedom."

Out-of-state speakers include Dr. Joseph C. Bunten, Cheyenne, president; Mr. John T. Flynn, New York, economist, author, and journalist; Dr. James L. Doenges, Anderson, Ind.; Marjorie Shearon, Ph. D., Washington, D. C.; Dr. William C. Black, San Diego; Dr. Edward Roland Annis, Miami; Dr. Richard Meiling, Washington, D. C.; and Leonard E. Reed, New York.

At the annual banquet Dr. Bunten will speak on "The Doctors' Security" and Mr. Flynn on "The Struggle of Our Time." Toastmaster will be Dr. Robert E. S. Young, Columbus, Ohio, immediate past president of the association.

Informal entertainment and social activities for women guests will be planned by members of the association from the host city. The annual meeting chairman is Dr. John H. Wootters, and Texas delegates to the association are Drs. T. C. Terrell, Fort Worth, John K. Glen, and Denton Kerr, Houston, and Leslie C. Powell, Beaumont.

Further information may be obtained from Mr. Harry E. Northam, Executive Secretary, 360 North Michigan Avenue, Chicago 1.

Radiologist Speaks to San Antonio Doctors

Dr. John C. Glenn, assistant professor of radiology at Duke University, Durham, N. C., spoke on "Radiologic Aspects of Diagnosis of the Gastrointestinal Tract" at a luncheon June 14 at Baptist Memorial Hospital, San Antonio, attended by members of the staff, announces the San Antonio *Express*. Dr. R. F. Gossett was program chairman.

Medical Board Examinations

The Texas State Board of Medical Examiners will hold examinations November 9-11 at the Blackstone Hotel, Fort Worth. Further information may be obtained from the secretary, Dr. M. H. Crabb, 1714 Medical Arts Building, Fort Worth.

AMERICAN COLLEGE OF PHYSICIANS COURSES

The American College of Physicians has planned nine postgraduate courses for fall of 1950 and winter of 1951. They will be given in Pittsburgh; Durham, N. C.; Chicago; New York; Salt Lake City; Rochester, Minn.; Philadelphia; and Boston. The schedule follows:

September 25-30, Pittsburgh, Internal Medicine, Selected Subjects, Dr. R. R. Snowden, Director.
October 9-14, Durham, N. C., Physiological Basis of Internal Medicine, Dr. Eugene A. Stead, Director.
October 23-27, Chicago, Critical Problems in Internal Medicine, Dr. Wright Adams, Director.
October 23-November 3, New York, Clinical Allergy, Dr. Robert A. Cooke, Director.
November 6-11, Salt Lake City, Recent Developments in Medicine, Dr. Max M. Wintrobe, Director.
November 27-December 2, Rochester, Minn., Peripheral Vascular Diseases Including Hypertension, Dr. Walter F. Kvale, Director.
December 4-9, Philadelphia, Gastroenterology, Dr. Henry L. Bockus, Director.
December 11-16, Boston, Selected Aspects of Clinical Hematology, Dr. William Dameshek, Director.
January 22-27, 1951, Philadelphia, Modern Trends in Diagnosis and Treatment of Heart Disease, Dr. William G. Leaman, Director.

Where facilities are available, courses will be open to nonmembers with adequate preliminary training; however, by direction of the Board of Regents, registrations from nonmembers of the College may not be accepted more than three weeks in advance of the opening of any course. The courses are no longer open to veterans under Public Law 346.

Additional information may be obtained by writing Mr. E. R. Loveland, Executive Secretary, 4200 Pine Street, Philadelphia 4.

UNIVERSITY OF TEXAS MEDICAL BRANCH

Chauncey D. Leake, Ph. D., vice-president and dean of the Medical Branch, has been appointed to direct the annual fund drive of the American Social Hygiene Association. Contributions to the fund will go toward programs which help parents prepare their children for marriage and family life and to investigate and report on commercialized prostitution throughout the country, states the Galveston *Tribune.*

Dr. George R. Herrmann has received an invitation to present a paper at the First International Congress on Internal Medicine, which will be held in Paris. He is also to present two papers for the First International Congress on Cardiology and will take part on the program of the centennial celebration of the University of Michigan Medical School, Ann Arbor, and the fourth triennial medical alumni conferences which will follow.

The following appointments have been made recently at the Medical Branch: Dr. William F. Spiller, clinical professor of dermatology and syphilology; Dr. Earl B. Ritchie, clinical professor of dermatology and syphilology; Dr. Mabel Wilken, associate clinical professor of neuropsychiatry; Dr. William A. Cantrell, assistant professor of neuropsychiatry; Dr. E. Ivan Bruce, assistant professor of neuropsychiatry; Dr. Garth L. Jarvis, assistant professor of obstetrics and gynecology; Dr. Gaynelle Robertson, associate professor of ophthalmology; Dr. Fred R. Guilford, assistant clinical professor of otorhinolaryngology; Dr. F. M. Townsend, associate professor of surgical pathology; Dr. D. D. Mark, assistant professor of pathology; Dr. Richard McDonald, assistant professor of pathology; C. W. McNutt, Ph. D., assistant professor of anatomy; Ethel C. E. McNeil, Ph. D., assistant professor of bacteriology and parasitology; John G. Bieri, Ph. D., assistant professor of biochemistry and nutrition; and Kenneth P. McConnell, Ph. D., assistant professor of biochemistry and nutrition.

A supplementary grant of $2,500 has been made by the Upjohn Company, Kalamazoo, to support studies on the use of antibiotics in surgery under the direction of Dr. Edgar J. Poth, Director of the Surgical Research Laboratory.

A supplemental grant of $3,000 has been made by the M. D. Anderson Foundation, Houston, to continue the support of studies on kidney pressure under the direction of Howard G. Swann, Ph. D., in the Physiology Laboratory.

Dr. Truman G. Blocker, Jr., professor of plastic and maxillofacial surgery, has received a special grant of $25,000 from the Army Medical Services for support of studies relating to thermal injury and epithelization.

A curriculum in physical therapy has been established at the Medical Branch under the medical direction of Dr. G. W. N. Eggers, professor of orthopedic surgery. The course leading to a bachelor's degree includes two years of general collegiate training and two years of special training in the new curriculum at the Medical Branch.

Lone Star State Medical Association Meets

The Lone Star State Medical Association composed of more than 100 Texas Negro physicians met June 13 to 15 in Galveston. Guest speakers were Dr. Joe Vincent Meigs, Boston, clinical professor of gynecology, Harvard Medical School; Dr. Alton Ochsner, New Orleans, professor of surgery, Tulane University; and Dr. M. H. Lambright, Cleveland, senior clinical instructor in Western Reserve University. Dr. Meigs spoke on cancer of the uterus; Dr. Ochsner addressed the association on phlebothrombosis. Dr. Lambright said to be the only Negro surgeon who does neurosurgery and all types of chest surgery, conducted surgical clinics, participated in a symposium on cancer of the lung, and spoke on the socialized medicine program of the Truman administration at a public meeting.

Other speakers and their topics were as follows: Drs. Truman G. Blocker, Galveston, the management and treatment of burns; Raymond Gregory, Galveston, hypertension; Willard Cooke, Galveston, ovarian cysts; John A. Wall, Houston, fibroid tumors; Jack E. Guinn, Fort Worth, intestinal obstruction; and William O. Russell, Houston. Dr. R. Lee Clark, Houston, led a session dealing with cancer problems.

Dr. A. L. Hunter, Marlin, took office as president of the association. Election of officers was as follows: Drs. F. E. Williams, Jr., Tyler, president-elect; H. L. Williams, Marlin, vice-president; M. L. Edwards, Hawkins, secretary; U. G. Gibson, Port Arthur, assistant secretary; and H. D. Patton, Palestine, treasurer. Chairman of the program committee for the meeting was Dr. J. M. Burnett, Fort Worth.

Austin was chosen as the site for the 1951 meeting to begin June 12.

New Director for State Hospital Board

Dr. Howard E. Smith, Sanatorium, medical director coordinator of Texas tuberculosis sanatoriums, has been appointed acting director of the State Board for Hospitals and Special Schools, states the Corpus Christi *Caller.* He succeeds Moyne Kelly who resigned as executive director of the board. Also resigning at the same time were W. C. Cason, business manager, and Dr. James Scarborough, Austin, clinical director of the board.

Northwest Texas Hospital Association

Members of the Northwest Texas Hospital Association will meet October 26 and 27 in San Angelo, according to the San Angelo *Standard-Times.* The oldest such organization in Texas, the association encompasses territory immediately west of Dallas, including Fort Worth, to San Antonio, and northwest to El Paso.

Southwest Poliomyelitis Center Opened

The Southwest Poliomyelitis Center was formally opened June 23 in Houston, reports the Houston *Chronicle*. The center is fourth of its kind in the United States.

Headed by Dr. William A. Spencer, the center occupies the entire tenth floor of Jefferson Davis Hospital. It is supported by the March of Dimes and operated by Jefferson Davis Hospital, the National Foundation for Infantile Paralysis, and Baylor University College of Medicine.

A $375,000 annex to Jefferson Davis Hospital is being completed this fall to house the center. It will have a capacity of 60 patients and will be equipped with modern facilities to handle the treatment of respiratory poliomyelitis patients. First the center will handle cases only in the Houston area; later it will serve not only Texas but also Louisiana, Arkansas, Oklahoma, and New Mexico.

PERSONALS

Dr. G. V. Brindley, Temple, immediate past president of the State Medical Association, on August 12 was elected chairman of the executive committee of the American Cancer Society, Texas Division, filling the vacancy created by the recent death of *Dr. E. W. Bertner,* Houston.

Dr. Alvis E. Greer, Houston, at the recent national convention of the American College of Chest Physicians in San Francisco, was named vice-president and regent of the College, reports the Houston *Chronicle.* He was presented an award of merit from the College for his outstanding work as president of the Southern chapter of the College. In addition, Dr. Greer has recently been elected chairman of the chest section of the American Medical Association.

Dr. Dudley Jackson, Sr., San Antonio, during July was awarded $4,885 for research work in cancer studies from funds made available by the National Cancer Institute, according to the San Antonio *Express.*

Dr. L. B. Zeis, Houston, has been elected to the board of directors of the Theatre Guild of Houston, reports the Houston *Chronicle.*

R. K. Clifton married *Dr. Jane Nash Creel* in Dallas recently, according to the University of Texas Medical Branch Alumni *Bulletin.*

A boy was born recently to *Dr. and Mrs. R. Sitta,* Chillicothe.

Recent parents of girls are *Dr. and Mrs. A. F. Johnson, Jr.,* Greenville; and *Dr. and Mrs. M. A. Caravageli* and *Dr. and Mrs. G. Willeford,* Galveston.

LIBRARY SECTION

PACKAGE SERVICE

The package library consists of collections of reprints and other periodical material on various subjects, prepared for lending to members of the Association. Request for packages should be addressed "Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." Twenty-five cents in stamps should be enclosed with the request to cover postage and part of the expense of collecting the material. Packages are allowed to remain in the hands of the borrower for 14 days.

ACCESSIONS

The following additions were made to the library during August:

Reprints received, 1,287.

Journals received, 304.

Books received. 23.

Techniques in British Surgery, Maingot; *Textbook of X-Ray Diagnosis,* Shanks, Cochrane, and Kesbey (editors); *Plastic and Reconstructive Surgery, Manual of Management,* Smith, W. B. Saunders Company, Philadelphia and London.

Practical Gynecology, Diagnosis, Treatment, Reich and Nechtow; *A.M.A. Council on Pharmacy and Chemistry, Reports, 1949,* and *New and Nonofficial Remedies, 1950,* Council on Pharmacy and Chemistry, A.M.A., J. B. Lippincott Company, Philadelphia.

Clinical Applications of Suggestion and Hypnosis, Heron; *Types of Diabetes Mellitus and Their Treatment,* Colwell; *Enzymes, Growth and Cancer,* Potter; *Oxygen Therapy,* Conroe and Dripps; *The Esophagus and Pharynx in Action,* Lerche; *Significance of Body Fluids in Clinical Medicine,* Newburgh, Charles C. Thomas, Springfield, Ill.

Scientific Principles in Nursing, McClain; *Professional Adjustments,* Lennon; *Management of Obstetric Difficulties,* Titus, C. V. Mosby Company, St. Louis.

Physicians Handbook, Krupp, Sweet, Jawetz, and Armstrong, 8th edition, University Medical Publishers, Palo Alto, Calif.

Malignant Disease, Treatment by Radium, Cade, 2nd edition, vol. 3; *Tumors of the Head and Neck,* Ward and Hendricks, Williams and Wilkins Company, Baltimore.

Nursery School Guide, Kellogg, Houghton Mifflin Company, Boston.

Stress, Treatise Based on Concepts of General Adaption Syndrome, Selye, 1st edition, Acta, Inc., Medical Publishers, Montreal, Canada.

The Antihistamines, Feinberg, Malkiel, and Feinberg, Yearbook Publishing Company, Chicago.

Cerebral Palsy, Pohl, 1st edition, Bruce Publishing Company, St. Paul, Minn.

SUMMARY OF SERVICE

Local users, 71.	Borrowers by mail, 51.
Items consulted, 1,775.	Packages mailed, 63.
Items borrowed, 353.	Items mailed, 252.

Film loans, 25.

MOTION PICTURE FILM LIBRARY

Motion picture films on medical subjects, 16 mm., both silent and sound, some in color, and suitable for either medical or lay audiences, are available for loan to county medical societies, hospital staffs, or individual physicians, on request. Borrowers will be required to pay only the cost of shipment of the films, by express, with insurance, and for any damage to films in the hands of the borrower.

Request for films should be addressed to "Motion Picture Film Library, State Medical Association of Texas, 700 Guadalupe Street, Austin, Texas." A list of available films, with descriptions, will be furnished on request.

The following motion picture films were loaned by the Film Library during August:

Anesthesia, Novocain, in Obstetrics (Winthrop Chemical Co.)—Mitchell Clinic, McKinney.

Anoxia, Physiology of (Linde Air Products)—Brackenridge Hospital School of Nursing, Austin.

Appraisal of the Newborn (Mead Johnson)—St. Paul's School of Nursing, Dallas.

As Others See Us (American Hospital Association)—Brackenridge Hospital School of Nursing, Austin.

Be Your Age (Metropolitan Life Insurance Company)—Brackenridge Hospital School of Nursing, Austin.

Breast Cancer: Problem of Early Diagnosis (American Cancer Society, Texas Division, Houston)—Cuero Hospital and Clinic, Cuero, and Dr. C. G. Goddard, Bastrop.

Bronchial Asthma (E. Fougera and Co.)—Brackenridge Hospital School of Nursing, Austin.

Coming Home (National and Texas Tuberculosis Association)—Dr. Robert W. Williams, Shiner.

Empyema, the Treatment of (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Extracellular Fluid (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Gastrectomy, Safer (Billy Burke Productions)—Dr. G. H. Brandau, Houston.

Goiter Surgery (Mead Johnson)—Dr. G. H. Brandau, Houston.

Hemicolectomy for Carcinoma of the Right Side of the Colon (Dr. Philip Thorek)—Dr. C. G. Goddard, Bastrop.

Hepatitis, Observation on (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Management of the Failing Heart (Varick Pharmacal Company)—Brackenridge Hospital School of Nursing, Austin, and Cuero Hospital and Clinic, Cuero.

Mechanism of Cell Division (U. S. War Department)—Veteran's Administration Hospital, Legion.

Middletown Goes to War (Texas Tuberculosis Association)—Shiner Rotary Club, Shiner.

Once Upon a Time (Metropolitan Life Insurance Company)—Dr. C. G. Goddard, Bastrop.

Pneumonia (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Post-Poliomyelitis Paralysis, Operative Procedures (National Foundation for Infantile Paralysis)—Veteran's Administration Hospital, McKinney.

Premature Infant, Care of (Mead Johnson)—St. Paul's School of Nursing, Dallas.

Strabismus Surgery (Dr. Ray K. Daily)—Rio Grande Valley Eye, Ear, Nose, and Throat Society, Harlingen.

Trichomonal and Monilial Vaginitis (G. D. Searle and Co.)—Mitchell Clinic, McKinney, and Scott and White Hospital School of Nursing, Temple.

Varicose Veins, Treatment of (G. D. Searle and Co.)—Brackenridge Hospital School of Nursing, Austin.

What Is Cancer? (American Cancer Society, Texas Division, Houston)—Cuero Hospital and Clinic, Cuero.

BOOK NOTICES

[1]Tumor Topics

R. Lee Clark, Jr., B. S., M. D., M. S. (Surgery), Director and Surgeon-in-Chief, The University of Texas, M. D. Anderson Hospital for Cancer Research; and Russell W. Cumley, B. A., M. A., Ph. D., The University of Texas, M. D. Anderson Hospital for Cancer Research, Houston, Editors. Cloth, 126 pages. $4. Houston, The Medical Press, 1950.

This little book includes a collection of articles which have appeared previously in the *Texas Cancer Bulletin*. They consist of brief, well written abstracts relating to various cancer subjects incorporating the most recent facts and figures of cancer specialists. The articles are arranged according to organ systems and consequently are readily available for reference. The book is recommended as a quick and ready means of obtaining pertinent information with regard to current thought on the treatment and diagnosis of cancer.

[1]*John F. Thomas, M. D., Austin.*

[2]You and Your Heart

H. M. Marvin, M. D., and others. Cloth, 306 pages. $3. New York and Toronto, Random House, 1950.

This volume is described as a clinic for laymen on the heart and circulation.

The reputations of the authors guarantee authoritative information and the book should be freely recommended to patients who seek further information about the heart and its diseases. Such a book is necessary, as so much questionable information is available in the various popular monthly magazines.

The section by Dr. Rutstein, "Public Health Aspects of Heart Disease," could serve as a guide for lay groups interested in learning reasons and techniques for the establishment of local social and rehabilitation services. It should also serve as a guide for those of the medical profession who do not understand the reasons for joint effort by private practitioners and organized public agencies.

The authors, with the help of Mr. David Loth, have achieved clarity and simplicity. The book can be profitably read by physicians and laymen.

[3]Trends in Medical Education

The New York Academy of Medicine Institute on Medical Education, 1947; edited by Mahlon Ashford, M. D. Cloth, 320 pages. $3. New York, The Commonwealth Fund, 1949.

This volume is a current reflection from medical leaders in the eastern United States concerned with the continuing problems of medical education, particularly as they relate to current social problems. In his address opening the institute Dr. George Baehr indicates the desire of conscientious medical administrators to coordinate medical facilities most effectively for the educational program.

The discussion is arranged with relation to (1) premedical education and selection of medical students, (2) undergraduate medical education, (3) medical education of interns and residents, (4) graduate and postgraduate training for specialization, (5) the responsibility of medical schools and hospitals in the education of the general practitioner, and (6) the relation of group practice and community medical services to medical education. While there is nothing new or startling to the alert medical educator in these discussions, much material in the volume would be of value to practicing physicians and to responsible lay leaders who are concerned with current problems of medical care.

As a profession medicine has always maintained the highest possible standards of service. This involves related high standards of education. It is to the credit of the medical profession that so many of its teaching methods have been adopted in general educational programs, such as the case history method and the preceptorial technique. Both are of extreme antiquity as far as medicine is concerned. It is important that medical schools continue to experiment with techniques and methods of training.

The discussions in this volume present the viewpoints of urban medical leaders, particularly those in the northeastern part of the United States. It does not reflect the experimental efforts in medical education that are being made in many other parts of the country, including such ideas as

[2]*E. Grey Dimond, M. D., Austin.*
[3]*Chauncey D. Leake, M. D., Galveston.*

preceptorial assignments of students with selected physicians in contiguous areas, the student-unit laboratory to provide greater correlation of preclinical and clinical studies, a continuing effort toward more logical realignment of curricular subjects, and seminar programs for advanced and graduate work in medicine.

Appropriately the volume closes with a consideration of the development of group practice and the reflection of this tendency in medical education. Medical students have every opportunity to learn the advantages of associated endeavor in medical schools, and particularly in the hospitals which are organized and developed for effective medical teaching. Group medical practice seems to be favored by economic circumstances, by convenience of effort, and by efficiency and effectiveness as far as individual sick patients are concerned. This volume may be important to those who are interested in developing postgraduate medical education in Texas.

[4]The Law of Medicine

Parnell Callahan, A. B., LL. B., Member of the New York Bar; and Justin Callahan, A. B., M. D., Assistant Attending Obstetrician and Gynecologist, New York Hospital; Instructor in Obstetrics and Gynecology, Cornell University Medical School; Assistant Attending Obstetrician and Gynecologist, St. Clare's Hospital; Junior Assistant Attending Gynecologist, Roosevelt Hospital. Paper, 80 pages. $1. New York, Oceana Publications, 1950.

"The Law of Medicine" is the twentieth publication in the Legal Almanac Series and presents a broad and general, nontechnical explanation of the law insofar as it relates to the practice of medicine. It is written in outline form, is easy to read, and as introductory, background material serves a useful purpose. It should not, however, as the publisher indicates, take the place of an attorney's advice on any specific legal problem.

[5]The Nose

Thomas H. Holmes, M. D., Lester N. Hofheimer Research Fellow in Medicine; and others. Cloth, 154 pages. $4.50. Springfield, Ill., Charles C. Thomas, 1950.

This short monograph of 154 pages is truly a new addition to the literature in otolaryngology. It might even be termed psychiatry in the practice of otolaryngology because it deals primarily with changes in the nose and respiratory passages which accompany certain emotional threats and upsets. The author gives in detail his method of study and observation in normal and pathologic subjects. Case histories showing various symptoms and clinical findings are given in such detail as to make them enjoyable and instructive reading, and mechanisms involved in disorders of the upper respiratory tract accompanying these various emotional changes are discussed in considerable detail. This volume is well worth being read and studied by the otolaryngologist or general practitioner, because it offers a satisfactory explanation for some of the nasal symptoms for which otherwise they are not able to find a cause.

[6]Clinical Uses of Intravenous Procaine

David J. Graubard, M. D., Assistant Visiting Surgeon, Cumberland Hospital; Assistant Visiting Orthopedist, Kingston Avenue Hospital, Brooklyn, N. Y.; and Milton C. Peterson, M. D., Visiting Anesthesiologist, Research Hospital, Kansas City, Mo.; Associate Professor of Anesthesia, New York Post-Graduate Medical School, New York. Cloth, 104 pages. $2.25. Springfield, Ill., Charles C. Thomas, 1950.

This readable little book (less than 100 pages) is evidently written by men who are primarily concerned in conveying information. It is a concise report on a considerable amount of work by many men in a small corner of the field of medicine. It should be of interest to surgeons, anesthesiologists, internists, and those in general practice.

First is a short historical sketch, then a brief outline of the scientific principles involved and techniques employed. Then is presented data (both favorable and unfavorable) on the intravenous use of procaine in a variety of conditions. In this technique is a tool not generally appreciated and one which is worth a wider trial in many traumatic and inflammatory conditions, allergic states, complications of anesthesia, poliomyelitis, and so forth.

[7]Intestinal Intubation

Meyer O. Cantor, M. S., M. D., F.A.C.S., Assistant Attending Surgeon, Grace Hospital, Detroit. Cloth, 333 pages. $7.50. Springfield, Ill., Charles C. Thomas, 1949.

The author has compiled a timely book on intubation of the gastrointestinal tract, a diagnostic and therapeutic procedure which has made marked advancements within the past decade. Included in the volume are a history of the development of intubation together with an anatomy and physiology of the gastrointestinal tract as refers to intubation.

A description of types of long intestinal tubes is given along with the indications, advantages, disadvantages, and techniques for their passage. All of the various types are considered as well as one of the author's own design.

The makeup of the book is good with many adequate illustrations. There is some repetition of material under various chapter headings, but it primarily emphasizes important points.

There is little need to refer to the extensive bibliography, since pertinent literature within the past ten years is well covered within the book. The book can be recommended without reservation to gastroenterologists and surgeons who are concerned with intubation.

[8]Transactions of the American Goiter Association 1949

American Goiter Association, Annual Session, Madison, Wisc. Cloth, 460 pages. $10.50. Springfield, Ill., Charles C. Thomas, 1950.

Papers presented at the annual meeting have been collected in a single volume. Frequently these dissertations have been reprinted elsewhere. Consequently only a review of a general nature is possible; it is beyond the scope of this review properly to discuss and evaluate individual articles. The importance of this type of compilation is that the current thought dealing with the normal and abnormal physiology of the thyroid is set forth in its various phases.

Great emphasis is placed on studies using the isotope iodine to follow reactions and physiologic processes in the gland and the fate of administered substances.

The treatment of hyperthyroidism is considered from the viewpoint of surgery, antithyroid drugs, and radiation both external and internal. Unfortunately, the dangers of internal radiation, using even the more unstable radioactive elements such as Iodine^{131}, are not sufficiently stressed. No mention is made of the damage which could result to the fetus of a pregnant woman receiving Iodine^{131} in sufficient quantities to retard the hyperplastic gland in Grave's Disease. From what is now known it is obvious that the therapeutic use of radioactive isotopes should be restricted to medical

[4]*Fred Niemann, LL. B., Austin.*
[5]*P. W. Malone, M. D., F.A.C.S., Big Spring.*
[6]*E. R. Huckleberry, M. D., Waller.*
[7]*John F. Thomas, M. D., Austin.*
[8]*Edgar J. Poth, M. D., Galveston.*

centers especially studying the problems involved. Their widespread use cannot as yet be justified.

A good section is given to benign and malignant tumors of the thyroid, but nothing startlingly new is presented.

The technique of round-table discussions is excellent and gives insight into several views on a subject, not merely the considered writing of one author. A study of such compilations is certainly to be recommended to all interested in this subject, whether or not they have been fortunate enough to have attended the meeting.

[9]Chemical Developments in Thyroidology

William T. Salter, M. D., Professor of Pharmacology, Yale University School of Medicine, New Haven, Conn. Fabrikoid, 87 pages. $2. Springfield, Ill., Charles C. Thomas, 1950.

This excellently written little monograph consists essentially of four essays dealing with: (1) synthesis of the thyroid hormone, (2) development of blocking agents which impede natural synthesis, (3) the nature of the peripherally circulating hormone, and (4) application of radioiodine to problems in physiology and therapy. It thus summarizes recent advances in the basic physiology of the thyroid gland which have played so important a role in our present concepts of the thyroid and its disorders. The book can be highly recommended to the reader desiring a brief resumé of the field.

It is to be regretted that the subject of thyroid physiology was not covered more extensively, the present volume being no longer than the average review article and limited to the chemical aspects of a subject which is of greatest importance to the physician interested in the proper management of thyroid diseases.

[10]Some Relations Between Vision and Audition

J. Donald Harris, Ph. D.; Head, Sound Research Section U. S. Naval Medical Research Laboratory, New London, Conn. Fabrikoid, 56 pages. $1.50. Springfield, Ill., Charles C. Thomas, 1950.

With the ultimate aim to acquire knowledge of the theory of the whole sensorium, the author sets out to gather information of the physiology of seeing and hearing. The result is not a complete textbook on all phases of the physiology of these higher senses but a fine collection of certain aspects of vision and audition. In about a dozen chapters the reader is introduced to many problems of perception and learns how much visual and auditory phenomena differ from and resemble each other. The author's review covers such subjects as sensitivity of the eye and ear, dark adaptation of the eye and auditory fatigue, the individual nerve fiber, central (cranial) perceptive acuity, and intersensory relations and facilitations.

The book is supported by a large and up-to-date bibliography. It will be welcome to anybody who is interested in physiology, experimental psychology, and related subjects.

[11]Water and Salt Depletion

H. L. Marriott, C.B.E., M. D., F.R.C.P., Middlesex Hospital, London, England. Fabrikoid, 80 pages. $2. Springfield, Ill., Charles C. Thomas, 1950.

This monograph consists of a series of lectures delivered by Dr. Marriott on water and salt depletion.

The various clinical and physiochemical changes occurring in water depletion, salt depletion, and water and salt depletion are discussed at length and the proper treatment for each is presented.

The mechanisms whereby the body attempts to maintain the isotonicity of the extracellular fluid are discussed in great detail.

This monograph is well written and is certainly worth reading.

[12]Diagnosis and Therapy of Gynecological Endocrine Disorders

C. L. Buston, M. D., Med. Sc. D.; Associate Professor of Clinical Obstetrics and Gynecology, Columbia University College of Physicians and Surgeons; Associate Attending Obstetrician and Gynecologist, Sloane Hospital for Women, New York; and E. T. Engle, Ph. D.; Professor of Anatomy, Columbia University; College of Physicians and Surgeons, New York. Fabrikoid, 62 pages. $2. Springfield, Ill., Charles C. Thomas, 1949.

As the authors say, this book is written for the general practitioner and internist. The brief review of the physiology of menstruation and the hormonal control of the menstrual cycle, as well as the systemic activity of the menstrual cycle, is excellent. The whole subject matter is well presented, being thorough but concise, without exhaustive rambling details. The diagnosis of each endocrine disorder is discussed thoroughly.

It is my opinion that this short book will be of more use to the general practitioner and internist than any written in the last few years.

[13]A Primer for Diabetic Patients

Russell M. Wilder, M. D., Ph. D., F.A.C.P., Professor and Chief of the Department of Medicine of the Mayo Foundation, University of Minnesota; Senior Consultant in the Division of Medicine, Mayo Clinic. Ninth edition. Cloth, 200 pages. $2.25. Philadelphia and London, W. B. Saunders Company, 1950.

The ninth edition of Wilder's "A Primer for Diabetic Patients" incorporates the newer dietary concepts. The author recommends more liberal feedings when insulin is used. He stresses the importance of a careful regimen; however, he attempts to teach the patient with diabetes how to attain the best possible health and vigor.

This concisely written handbook of 200 pages, primarily for the diabetic patient, gives careful instructions in the cause, treatment, and complications of the disease. Each chapter is followed by a series of questions which tend to emphasize the most important features covered. The diabetic patient of average intelligence can study the book and attain an excellent understanding of his disease. Attached to the back cover is a food nomogram which is not too complicated and which helps the patient determine his basic food requirements. It should be invaluable to any physician who treats diabetes mellitus.

The patient should be allowed to study the book thoroughly thereby gaining the basic knowledge of his disease. His care then will be much simpler for the physician, and the patient will lead a much safer and happier life.

[14]The 1949 Year Book of Pathology and Clinical Pathology

Howard T. Karsner, M. D., LL. D., Medical Research Advisor, Bureau of Medicine and Surgery, United States Navy; and Arthur Hawley Sanford, M. D., Professor of Clinical Pathology, University of Minnesota (The Mayo Foundation); Emeritus Consultant, Division of Clinical Laboratories, Mayo Clinic, Editors. Cloth, 543 pages. $4.75. Chicago, The Year Book Publishers, 1950.

In a half century of annual publications these books have established a justifiably excellent reputation. Their purpose is threefold: to present the new, evaluate the recent, and discard the old. In the 1949 edition the usual high standards and continuity are maintained. Slightly less than half the

[9]*Arthur Grollman, M. D., Dallas.*
[10]*Otto Lippmann, M. D., Austin.*
[11]*George Clifford Thorne, M. D., Austin.*
[12]*Doyle L. Patton, M. D., Midland.*
[13]*A. O. McCary, M. D., Freeport.*
[14]*John J. Andujar, M. D., Fort Worth.*

book is devoted to clinical pathology and the remainder to anatomic pathology, with an all too brief group of appendices, one of which is an excellent review of advances in the manufacture of apparatus during 1949.

Some perennial theses are given their annual airing. Thus, Frank Foote's excellent paper proving aberrant thyroid to be metastatic thyroid cancer again is given the full treatment, with illustrations. The old argument on nomenclature of basal cell tumors is also resurrected, quoting the new term "basalioma." Even in doing so, however, the editors proclaim that they include such articles to illustrate "the introduction of philosophy in the thinking of pathologists." Amen, say we!

New contribution on paper chromatography, the Carter Rh haptens, and recent contributions on air embolism (which probably will be carefully read in New England), together with a long chapter on recent advances in cytology, round out the book.

Pathologists and laboratorians who wish to keep up with the enormous strides of scientific medicine will want to add this latest edition to their collection of year books.

[15]The 1949 Year Book of Endocrinology, Metabolism and Nutrition

Willard O. Thompson, M. D., Clinical Professor of Medicine, University of Illinois College of Medicine; Attending Physician (Senior Staff), Henrotin Hospital; Attending Physician, Grant Hospital of Chicago; and Tom D. Spies, M. D., Chairman, Department of Nutrition and Metabolism, Northwestern University School of Medicine; Director, Nutrition Clinic, Hillman Hospital, Birmingham, Ala., Editors. Cloth, 550 pages. $4.75. Chicago, The Year Book Publishers, Inc., 1950.

From the standpoint of the endocrinologist, 1949 has been a banner year. Cortisone and ACTH have caused the profession in general to give considerable thought to the possibility that many concepts of disease etiology will have to be radically revised. Dr. Thompson has selected outstanding contributions which are beneficial to the profession as a whole.

Nutrition has come to the front as a science. In order that the busy practitioner may have accessible scientific data regarding this important problem, the volume makes this information available in a concise and accurate manner.

[16]Textbook of Bacteriology

Joseph M. Dougherty, A. B., M. A., Ph. D., Dean of the School of Science and Professor of Bacteriology, Villanova College; Fellow of the American Association for the Advancement of Science; and Anthony J. Lamberti, B. S., M. S., Instructor in Bacteriology and Parasitology, Temple University School of Medicine. Second edition. Cloth, 491 pages. $5.75. St. Louis, C. V. Mosby Company, 1950.

This new edition brings the book generally up to date. Additional attention has been given to bacterial types. Effects of physical and chemical agents, including the "wonder drugs," on bacteria receive new treatment. Instances occur where up-to-dateness falls a trifle short, such as neglect to bring Bergey classification names up to the last revision, but they are of little consequence.

The presentation is primarily for students in medical and related professional courses. Premedical students like to take bacteriology to simplify the stiffer medical bacteriology ahead, though it involves going over some of the same ground twice. The material in the book is adequate for such purpose and is readibly presented. At places the sequence seems illogical, as when the discussion of bacteria is dropped for the consideration of fungi and then resumed again, but this is not a serious impairment.

The book is intended for general college bacteriology as well as premedical bacteriology. It accepts the traditional trend of college bacteriology to overemphasize medical aspects at the expense of nonmedical aspects. Water, dairy, and other fields in industrial and agricultural bacteriology, have much to interest an enlightened public, yet only a chapter is devoted to water, milk, and food. It is true that few college texts do more. However, the reviewer would like to see a college course which rather than abbreviate medical bacteriology would relate all of the several roles that bacteria play in this life we live.

[17]Anxiety in Pregnancy and Childbirth

Henriette R. Klein, M. D., Associate in Psychiatry, Columbia University College of Physicians and Surgeons, New York, and others. Cloth, 111 pages. $2.75. New York, Paul B. Hoeber, Inc., 1950.

The authors have gone into rather careful detail in studying twenty-seven pregnant women throughout pregnancy and postpartum, analyzing their anxieties and general reactions to pregnancy and delivery. The study was carried out with a rather unstable group in general, and during the period of difficult housing following the war. The book emphasizes the importance of the psychosomatic in obstetrics.

[18]Medical Gynecology

James C. Janney, M. D., F.A.C.S., Associate Professor of Gynecology, Boston University School of Medicine. Associate Visiting Gynecologist, Massachusetts Memorial Hospitals. Second edition. Cloth, 454 pages. $6.50. Philadelphia and London, W. B. Saunders Company, 1950.

The second edition of Janney's "Medical Gynecology" is well arranged, easily read, and shows considerable improvement over the first edition. The book is divided into seven parts, making reading, review, or reference easily accomplished.

The first part is a short but adequate discussion of the important points to be noted in the history and physical examination and gives a good method for classification of the patient's condition.

Presentation of the patients' complaints in Part 2 is unusually well written. Under each separate complaint are listed the different physiologic and pathologic causes; a summary of differential points of importance in the history, physical examination, and laboratory findings; and conventional methods of treatment.

Part 3 consists of a small section devoted to the physical findings in different gynecologic conditions and adds little to that found in other gynecology textbooks. Part 4 gives fairly briefly the meaning of the various laboratory tests applicable in gynecology; apparently it is not intended to cover the technique of performing all the procedures discussed.

Most of the chapters in Part 5 on gynecologic treatment are well written and are valuable for review. Sufficient space has not been devoted to the chapters on endocrine therapy, antibiotic drugs, and chemotherapy in view of the many recent advances in these fields.

The subject of sociomedical problems is dealt with in an excellent manner in Part 6. Much helpful advice is given on premarital and marital problems and to problems of maladjustment.

Part 7 on radiation or operation is brief, summarizing the usual textbook opinions.

This book is highly recommended to medical students; it will also be of interest to physicians who wish to review.

[15]*Lester Conrad Feener, M. D., F.A.C.P., El Paso.*
[16]*William B. Sharp, M. D., Galveston.*
[17]*Celso C. Stapp, M. D., El Paso.*
[18]*Virgil C. Baxter. M. D., Galveston.*

ORGANIZATION SECTION

STATE MEDICAL ASSOCIATION

EXECUTIVE COUNCIL DECISIONS

Action relating to the channeling of necessary medical officers into military forces for service during the Korean emergency was the chief business transacted by the Executive Council of the State Medical Association when it met August 24 in Austin. Originally scheduled to meet in September, the Council convened at the earlier date because of the urgency of securing reserve medical officers.

With Dr. William M. Gambrell, Austin, President of the Association, presiding, members of the Executive Council and the specially invited presidents and secretaries of county medical societies heard Col. David E. Liston, San Antonio, surgeon of the Fourth Army, explain that despite the cooperation civilian physicians are giving in examining Army inductees, the combat demands of the Korean fighting necessitated the call of 38 Texas physician reservists by September 7 unless the quota could be filled by volunteers. It was pointed out that Public Law 365, Eightieth Congress, provides for a $100 per month bonus to medical officers who volunteer for active service. It was the hope of Army officials that the first call for 38 medical officers from Texas might be filled by volunteers who would be eligible to receive the $100 bonus.

Dr. F. J. L. Blasingame, Wharton, member of the Board of Trustees of the American Medical Association, told of the establishment by the A.M.A. four years ago of a Council on National Emergency Medical Service which has made numerous recommendations to the Department of Defense relating to the conservation of medical manpower and other pertinent problems. American Medical Association officials, realizing that the Korean situation would require additional medical officers, attempted to encourage the volunteering of young physicians who were educated at government expense or were deferred from service during World War II to complete their medical studies. Sufficient volunteers were not forthcoming, and it became apparent that draft legislation would be the only method of calling the group of physicians mentioned. The Board of Trustees of the American Medical Association therefore urged the passage of legislation which would provide for drafting physicians according to the following priority:

1. Former students in the A.S.T.P. or V-12 programs and persons deferred during World War II in order to continue their medical education and who have had no active duty.

2. Those deferred previously for other than physical disability and those previously rejected for physical disability who may be found fit on the basis of present physical requirements.

3. Former students in the A.S.T.P. or V-12 programs who served on active duty as commissioned officers for less than 21 months (exclusive of time in postgraduate training).

4. Those who served less than ninety days on active, honorable military or naval duty.

5. Those whose total active, honorable military or naval duty is less than twenty-one months.

Legislation embodying these provisions has been introduced into Congress, but it was pointed out by Colonel Liston and others that military requirements for medical officers immediately could not wait for the passage and implementation of such bills.

Dr. R. A. Trumbull, Dallas, chairman of the State Council on National Emergency Medical Service, reported on two meetings in July held by his committee with representatives from the Fourth Army, primarily to work out a plan of cooperation between civilian physicians and the Army to assure physical examination of inductees with the least possible dislocation of physicians.

Dr. Everett C. Fox, Dallas, chairman of the Council on Medical Economics, reminded that unintentionally conflicting directives by the House of Delegates had left in question the proper committee to handle procurement and assignment problems in the event of an emergency such as now obtains. At the suggestion of Dr. Fox and upon the recommendation of a reference committee appointed to consider the problem, the Executive Council adopted a resolution to place matters of procurement and assignment of physicians in the hands of a Council on National Emergency Medical Service to be composed of the members of the Council on Medical Economics and the presently existing State Council on National Emergency Medical Service, the chairman to be appointed from this group by the President, and additional members to be appointed by the President so as to assure adequate geographical representation.

President Gambrell immediately complied with the resolution, and the Council on National Emergency Medical Service was constituted as follows: Drs. R. A. Trumbull, Dallas, chairman; Ozro T. Woods, Dallas; Glenn D. Carlson, Dallas; J. L. Goforth, Dallas; Hamilton Ford, Galveston; W. H. Hamrick, Houston; Everett C. Fox, Dallas; H. H. Cartwright, Breckenridge; E. W. Jones, Wellington; Tom B. Bond, Fort Worth; Raleigh R. Ross, Austin; R. B. G. Cowper, Big Spring; R. E. Windham, San Angelo; J. L. Cochran, San Antonio; Troy A. Shafer, Harlingen; and L. C. Powell, Beaumont.

The Executive Council voted that all information concerning military affairs from any source should be channeled through the central office of the State Medical Association to the individual members concerned and to the president and secretary of each component county medical society.

A resolution endorsing the draft legislation recommended by the Board of Trustees of the American Medical Association was adopted, and it was voted that a copy of the resolution should be sent to the President of the United States and to Senators and Congressmen from Texas.

Another resolution, introduced by Dr. Van Doren Goodall, Clifton, and unanimously adopted by the Executive Council, urged that the medical departments of the various military services consult with the Council on Medical Service of the American Medical Association on the necessary doctor-patient ratio in the armed services and that waste of medical officers and medical services be prohibited as far as possible.

It was requested that county medical societies establish procurement and assignment committees at once. The Executive Council passed a motion that the executive board of the county society appoint the county procurement and assignment committee and that where no such board exists the county society president appoint the committee. It was requested that the county committees proceed immediately to supply the Executive Secretary of the State Medical Association with the name of and other pertinent data about reserve medical officers commissioned in any of the armed services

and residing in the county society area, regardless of whether or not such officers are members of the medical society. (Although requests have come thus far from only the Army, it is anticipated that the other armed services, especially the Air Force, may require additional medical officers.) It was pointed out that lists of reserve officers furnished by the Texas Military District were incomplete and inaccurate. The county committees were also requested to notify the Executive Secretary by August 31 of the names of any physicians willing to volunteer. It was presumed that the county committees will be called on later for additional information.

Upon motion by Dr. Merton M. Minter, San Antonio, the Executive Council adopted the following procedure for handling requests for medical officers: The armed forces shall notify the central office of the State Medical Association of the necessary military requirements for physicians from Texas and these requirements shall be passed on to the Council on National Emergency Medical Service. This Council shall decide what method and in what manner these requests will be channeled to the county societies. When requests for medical officers are received by a county society president, the county procurement and assignment committee shall select from the society membership sufficient and suitable men to fill the request. Each physician so selected shall be notified by the society president, who at the same time shall notify the Council on National Emergency Medical Service of the physician's selection. If the man selected objects, he may be heard by the Council on National Emergency Medical Service or by a sub-committee of the Council consisting of not less than five men, none of whom shall be in his councilor district. If the selection, after appeal, is approved by the Council, appeal by the man selected shall be directed to military channels.

A number of items of business not connected with the military problem were considered by the Executive Council. Dr. Jim Camp of Pecos, elected in May by the House of delegates as general practitioner of the year, was presented a plaque in recognition of the honor. The Council voted to designate the dates of May 4-7 for the 1952 annual session in Dallas. The Council passed a motion to recommend to the House of Delegates that the By-Laws of the Association be amended to provide for semi-annual regular meetings of the Executive Council in January and September of each year, exact dates to be set by the President. Reports from the Board of Councilors, Council on Legislation, Committee on Public Relations, and several other committees were presented, and the activities of the Texas League for Health Education were outlined.

DR. JIM CAMP, GENERAL PRACTITIONER

A plaque proclaiming him as the general practitioner of 1950 in Texas was presented to Dr. Jim Camp of Pecos during the special meeting of the Executive Council of the State Medical Association in Austin on August 24. Dr. Camp, unanimously nominated by the physicians of District 1, was elected by the House of Delegates at the annual session of the State Medical Association last May to represent Texas as a candidate for the American Medical Association's award to the General Practitioner of the Year. Selection of the physician to receive the national honor will be made at the clinical session of the American Medical Association in December.

Dr. Camp was born at Doyle Station, White County, Tenn., November 7, 1877, the son of a farmer. He worked on the farm to help pay his way through high school and through Onward Seminary at Doyle Station. He was not graduated from either school but received from Onward a teacher's certificate, which he put to use immediately by teaching school for a year or two. During this period he studied medical books in his spare time, and in 1897 he enrolled at the University of Tennessee, Nashville. Upon his graduation in medicine in 1900, Dr. Camp went to Pecos, where some relatives operated a drug store, and began practice.

Throughout his fifty years of general practice in Pecos and the western Texas and eastern New Mexico area surrounding it, Dr. Camp has kept pace with changing methods of medical and surgical practice. He started with few instruments and aids, depending on a bicycle, horses, and freight trains to get him to his patients. By 1915 he had converted a house into a small hospital. This first hospital has evolved into a modern, well-equipped, specially-built structure in which the newer developments in medicine, which Dr. Camp has learned in annual postgraduate study in medical centers throughout the country, can be applied.

Although the practice of his profession has been the paramount interest in Dr. Camp's life, he has found time to contribute to his community in other endeavors. He was a

DR. JIM CAMP

member of the school board for twenty-five years, ten as chairman. He was instrumental in securing the establishment of the Red Bluff Water District and served as president of the Reeves County Water Improvement District 2. He acted as a vigilante in early law enforcement days and is still an active member of the Sheriff's Posse. He was a member of the County Democratic Executive Committee for many years and served as its chairman. In 1910 he served an elective term as county tax assessor. He and the late W. W. Dean built the Camp-Dean office building in downtown Pecos. Dr. Camp is a member of the Christian Church, served ten years as a deacon, and has served twenty-five years as an elder, participating in state-wide laymen's activities. He became a Master Mason soon after his arrival in Pecos and has held all offices in the Blue Lodge and chapter and is a member of the commandery. He is also a member of the Knights of Pythias and Woodmen of the World. He is a charter member of the Pecos Rotary Club, a member of the Chamber of Commerce, and a charter member of the Pecos Valley Country Club. He helped to organize the Richard Briscoe Post 91 of the American Legion and was its first commander; he had volunteered for service in World War I and was with the Army Medical Corps from June until December, 1918. He was given an emergency furlough from the Army shortly

before his discharge to help subdue an influenza epidemic in Pecos.

Dr. Camp early became affiliated with the county, district, state, southern, and national medical associations, and he has served several terms as president of the Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Medical Society. For many years he has been local surgeon for the Texas and Pacific and Santa Fe Railways. He is district surgeon for the Texas Highway Department and surgeon for the federal jail at Pecos.

Sharing the honors which have come to Dr. Camp is his wife, the former Miss Bird Stroud of Doyle Station, Tenn., who came to Texas to become his bride October 1, 1901. He has one son, Keith Camp, and one daughter, Mrs. Robert Dean, living in Pecos. A second son, Dr. Hilliard Camp, who practiced medicine with his father, was accidentally killed in 1941. Dr. Camp has established a scholarship at Texas Christian University, Fort Worth, in memory of this son.

COUNTY SOCIETIES

Bell County Society

June 7, 1950

(Reported by E. O. Bradfield, Secretary)

Changing Concepts of Diseases—R. G. Greenlee, Temple.

Sixty-four members and guests were present at the June 7 meeting in Temple of Bell County Medical Society. The committee on resolutions presented a resolution on the death of Dr. Charles M. Simpson, which was read by the secretary in the absence of the members of the committee. The resolution was read and adopted.

A letter from the president of the Temple Public School Board asking for recommendations of the society in regard to health certificates for school children after illness was read by Olin B. Gober. A committee was appointed to study the problem and report at the August meeting.

A. W. Sommer, Temple, was elected unanimously to membership.

The society voted to have the president appoint a successor to the chairman of the committee on public relations who is no longer in Temple and decided that otherwise the committee should continue to function for the remainder of the year. A. Ford Wolf reported on the past annual session of the State Medical Association.

Brazoria County Society

June 29, 1950

(Reported by W. T. Galloway, Secretary)

Hydronephrosis (lantern slides)—Howard T. Fretz, Wharton.

The Brazoria County Medical Society held its regular meeting June 29 at West Columbia, and the program outlined above was given. The committee on insurance policies reported.

Collin County Society

June 12, 1950

At the June 12 meeting of Collin County Medical Society a charter was reissued to the society. Frank Selecman, Dallas, district councilor, was present.

Dallas County Society

June 13, 1950

(Reported by W. W. Fowler, Secretary

Evaluation of Routine Cytologic Studies of Carcinoma of Cervix—Richard L. Hermes, Dallas.

Noninvasive Carcinoma of Cervix—A. J. Gill, Dallas.

Surgical Treatment of Genital Carcinoma—William F. Mengert, Dallas.

Discussion—G. F. Goff and W. P. Devereux, Dallas.

Elliott Mendenhall, president, presided at the June 13 meeting in Dallas of Dallas County Medical Society. The program outlined above was presented. Elected to membership were the following: Ben M. Colletti, Max Paul Goodfried, Jack Elwood Lanier, Powell Bower Loggan, and Clifford Charles Seidel. Transfer of memberships for Joseph P. McNeill, Galveston County Medical Society, and Elmer Russell Hayes, Hennepin County (Minnesota) Medical Society, were announced.

The chairman of the executive council, George Schenewerk, made the following report: Upon the report of the public health committee, the council recommended that the society go on record as being opposed to the principles involved in the proposal that the State Health Department establish a heart clinic in Dallas with funds furnished by the U. S. Public Health Service. The society voted to follow the recommendation of the council.

Members voted to postpone for action until the September meeting the recommendation of the council that Negro physicians in good standing in their own local medical organization be invited to attend scientific programs of the society. It was decided not to hold July and August meetings.

Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Society

June 15, 1950

A barbecue supper was given in Midland on June 15 for members of Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Medical Society by members of the auxiliary from Midland.

Gonzales County Society

August 9, 1950

(Reported by James C. Price, Secretary)

With two guests present the Gonzales County Medical Society had a dinner meeting August 9 at the home of N. A. Elder, Nixon. Dr. Elder gave a report on the annual meeting of the American Medical Association in September, which he attended.

Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Society

June 20, 1950

(Reported by Joe R. Donaldson, Secretary)

Indications for Pulmonary Resections—Francis J. Kelly, Amarillo.

The Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society met June 20 in Pampa and the program outlined above was given. Twenty members and four guests were present.

Dr. Kelly's talk included brief discussions of carcinoma, adenoma, suppurative diseases, cysts, and tuberculosis and was illustrated by several roentgenograms.

Robert Joseph Miller, Phillips, was elected to membership in the society, and J. J. Davis, Higgins, and H. W. Finley, McLean, were elected to honorary membership. A letter from Group Hospital Service, Inc. welcoming the society into the organization was read. Another letter from the State Board of Health stated that its facilities for chest roentgen-ray surveys would be available again this year. A leaflet from the State Tuberculosis Sanatorium which gave new rules for admission, length of hospitalization, and so forth, was read.

A golf tournament, a banker's handicap, was tentatively scheduled for July 30 in Pampa.

July 18, 1950

(Reported by Joe R. Donaldson, Secretary)

Fractures of Hip—John J. Hinchey, San Antonio.

Dr. Hinchey presented a paper, "Fractures of the Hip," at the July 18 meeting of Gray-Wheeler-Hansford-Hemp-

hill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society.

LaSalle-Frio-Dimmitt Counties Society

June 20, 1950

Members of LaSalle-Frio-Dimmitt Counties Medical Society and Auxiliary held their regular quarterly meeting in Dilley on June 20.

Lubbock-Crosby Counties Society

August 1, 1950

(Reported by Frank Hudgins, Secretary)

Modern Trends in Thoracic Surgery—F. J. Kelly, Amarillo.
Discussion—Albert G. Barsh, Forrest Freeman, and Julius T. Krueger, Lubbock.

Members of Lubbock-Crosby Counties Medical Society were guests of the Reese Air Force Base Hospital at a dinner and business meeting August 1, at which Roy G. Loveless, president, presided. Approximately sixty-five members and guests attended.

A letter from the Lubbock County Tuberculosis Association asking the society to participate in a clinic for indigent patients was read. Maurice J. Healy was accepted to membership by transfer from Mobile County (Alabama) Medical Society.

The president read a letter from the president of the State Medical Association which recommended that a procurement and assignment board and a military affairs committee be appointed.

Nueces County Society

June 13, 1950

(Reported by F. W. Yeager, Secretary)

Feeding the Hospitalized Patient (motion picture)—Courtesy of E. R. Squibb and Sons.

Fifty-three members attended the June 13 meeting in Corpus Christi of Nueces County Medical Society. L. M. Garrett reported concerning courthouse records. Sam Powell stated that during the past two years twenty-one deaths in the county had been caused by tubercular meningitis. He moved and W. R. Metzger seconded, that the public health committee make a study as to whether or not B.C.G. should be used for preventive purposes in the community.

A communication from the City Council of the Parent Teachers' Association commending the society for its cooperation in the preschool round-up was read.

E. Jackson Giles, president, read a letter from the executive board of Community House asking the society's approval for its project of obtaining garbage cans and screening for areas where there is infant diarrhea. After a general discussion the society voted to approve the project.

J. M. Cameron, Bette Upshaw, and J. E. Upshaw were admitted to the society upon application. Paul Gray reported for the committee which was appointed to investigate Veterans Administration practices. He said that the American Medical Association has set up a similar committee which is in the formative stage. He also stated that veterans who served in the occupation forces or other duty in peacetime could be treated only for service connected disabilities.

Elizabeth Thomason reported for the public health committee regarding information requested by the Chamber of Commerce about poliomyelitis in Nueces County. She also reported recommendations of the committee for the improvement of public health in the county. After much discussion it was decided to send the Chamber of Commerce two letters, the first answering the query as to the status of poliomyelitis in the county and the second dealing with the public health recommendations, namely, (1) the manner in which poliomyelitis cases should be reported by the newspapers, and (2) the provision of adequate sewers and storm sewers for all parts of the city in an effort to eliminate open toilets and cesspools.

Pecos-Jeff Davis-Presidio-Brewster Counties Society

July 11, 1950

(Reported by W. E. Lockhart, Secretary)

Allergy—G. H. Wood, Big Spring.
Crow's Nasopharyngeal Radon Applicator in Treatment of Pharyngeal Tonsils—G. H. Wood, Big Spring.
Radon Treatment of Vernal Catarrh—H. J. Roberts, Big Spring.

The Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society met July 11 at Iraan with six members and three guests present.

A letter from George Turner, El Paso, district councilor, relative to a school concerned with atomic energy problems was read and discussed. The motion was made by C. E. Oswalt, Jr., Fort Stockton, and seconded by Ben A. McReynolds, Fort Stockton, that the name of William D. Petit, Presidio, be proposed for honorary membership at the next annual session. The motion carried unanimously.

A letter from Malone V. Hill to the International Boundary Commission regarding work for the federal government was read.

August 1, 1950

(Reported by W. E. Lockhart, Secretary)

Pediatric Urology—H. M. Gibson, Jr., El Paso.
Head Injuries—W. A. Jones, El Paso.

Nine members and three guests attended the August 1 meeting in Fort Stockton of Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society. The program outlined above was presented by W. H. Stover, Marfa, who was in charge of the program.

Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Society

May 25, 1950

Orthopedic Problems—S. P. Rogers, El Paso.

Dr. Rogers, orthopedist, spoke at the May 25 meeting in Pecos of Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Medical Society. The auxiliary met with the society for dinner, after which separate meetings were held.

San Patricio-Aransas-Refugio Counties Society

June 21, 1950

L. G. Wood was host at a dinner meeting of San Patricio-Aransas-Refugio Counties Medical Society in Rockport on June 21. R. R. Lang, Corpus Christi gynecologist, addressed the society.

Titus, Morris, and Camp Counties Societies

May 29, 1950

Radiologic Helps in Modern Medical Practice—J. R. Maxfield, Dallas.

About thirty persons, including physicians' wives, attended the triennial meeting May 29 in Mount Pleasant of the Titus, Morris, and Camp Counties Medical Societies. In connection with his speech Dr. Maxfield showed three motion pictures, "Tale of Two Cities," "One World or None," and "What Atomic Energy Means to You."

Tom Green-Eight County Society

June 5, 1950

(Reported by Perry J. C. Byars, Secretary)

Malpractice Problems and Their Prophylaxis—Mr. Mark M. Myers, Dallas.

The regular monthly meeting of Tom Green-Eight County Medical Society was held at Sanatorium on June 5 after a dinner at the McKnight State Sanatorium. Thirty-two members attended the dinner and at the meeting afterwards the staff of the sanatorium were guests of the society.

Mr. Myers, representative for the Medical Protective Association, discussed the subject given above. A general discussion followed.

James N. White, delegate to the State Medical Association, gave a report of the annual session in Fort Worth. R. E. Johnson reported on the recent tuberculosis survey held in San Angelo. Upon motion by W. D. Anderson and second by S. H. Martin the society voted to extend an invitation to the Texas Tuberculosis Association to conduct an annual mass survey in San Angelo.

C. A. Kunath reported for the committee on indigent care in San Angelo and Tom Green County. After considerable discussion W. D. Anderson moved and R. E. Windham seconded that the report be sent to the Community Council, assuring the society's full support in the council's attempts to have the city and county underwrite the medical care of indigents in the locality. The motion carried, with one member, Harvey M. Williams, dissenting.

Letters from the State Medical Association regarding the President's Reorganization Plan 27 and Senate Resolution 92 were read. T. G. Coleman moved and S. H. Martin seconded that the society extend an invitation to the State Crippled Children's Association to hold its annual clinic in San Angelo this year, and the motion carried unanimously.

A discussion of discrepancies in the local telephone directory then took place, and the matter was referred to the board of censors to study and then discuss with the telephone company.

DISTRICT SOCIETIES

Seventh District Society

(Reported by John F. Thomas, President)

A chicken dinner and business session were held July 20 in Bastrop State Park by Seventh District Medical Society. Approximately 100 members, wives, and guests were present.

John F. Thomas, Austin, was elected president; A. J. Rice, Georgetown, vice-president; and G. W. Tipton, Austin, secretary-treasurer. Raleigh Ross, Austin, chairman of the legislative committee of Travis County Medical Society, discussed the qualifications of candidates for state office in the Democratic primary.

Twelfth District Society

July 11, 1950

(Reported by Nellins C. Smith, Secretary)

MORNING

Prefrontal Lobotomy with Report of Cases (motion pictures)—William L. Crosthwait, Waco.
Pulmonary Complications of Shock—R. R. Shaw, Dallas.

AFTERNOON

Coronary Insufficiency and Its Management—Howard Heyer, Dallas.
Fractures of Tibia—Herbert E. Hipps, Waco.

The Twelfth District Medical Society met July 11 at Cleburne and the program outlined above was presented. William M. Gambrell, Austin, president of the State Medical Association, spoke at the noon luncheon which was attended by members of the Woman's Auxiliary. The next meeting of the society will be January 9, 1951, at Temple.

Thirteenth District Society

October 10, 1950

(Reported by S. W. Wilson, Secretary)

Recent Developments in Use of Atomic Energy—R. S. Clayton, Dallas.
Obstetrical Problems—Garth L. Jarvis, Galveston.
Problems of Fluid Balance—Carl Moyer, Dallas.

Among the papers to be presented at the October 10 meeting in Mineral Wells of the Thirteenth District Medical Society are the three listed above.

Fourteenth District Society

June 13, 1950

(Reported by L. W. Johnston, Secretary)

MORNING

Malcolm Johnson, Paris, and W. P. Philips, Greenville, Presiding
Uretero-Pelvic Obstruction—John Shea, Gainesville.
Use of Tantalum Mesh in Hernia Repair—T. H. Thomason, Fort Worth.
Legislation of Concern to the Medical Profession—Mr. Philip R. Overton, Attorney, and Mr. W. E. Syers, Public Relations Counsel, State Medical Association, Austin.
Cancer of Face, Mouth, and Neck—Charles L. Martin, Dallas.
Remarks—Frank Selecman, Councilor of Fourteenth District, Dallas.

AFTERNOON

B. C. Wallace, Jr., Waxahachie, and V. C. Cirone, Gainesville, Presiding
What American Academy of General Practice Means to You—Frank Daugherty, Fort Worth.
Acute Pericarditis Simulating Coronary Occlusion—W. J. Gleckler, Sherman
Cortisone and ACTH—Arthur Grollman, Dallas.

Fifty-two members of the Fourteenth District Medical Society attended a one-day scientific program and business session June 13 in Paris. A meeting of the Woman's Auxiliary was held concurrently.

Mayo Tenery, Waxahachie, was elected president; N. L. Barker, Paris, vice-president; and L. W. Johnston, Terrell, secretary-treasurer. It was agreed to meet next in June, 1951, in Bonham.

Dr. William M. Gambrell, Austin, President of the State Medical Association, spoke at a luncheon for physicians and their wives given by Lamar County Medical Society and presided over by Drs. J. Shirley Sweeney, Gainesville, and Johnston. The Lamar County Society and Auxiliary were also hosts at a picnic in the evening.

The scientific program which was given is outlined above.

AUXILIARY SECTION

AUXILIARY NEWS

Twelfth District Auxiliary

The Woman's Auxiliary to the Johnson County Medical Society was hostess July 11 in Cleburne to members of the Twelfth District Auxiliary, which met concurrently with the district medical society. Approximately thirty-one members attended.

Mrs. John E. Talley, Waco, district council woman, presided at the business session. Guests received door favors of bottles of cologne in the center of ribbon nosegays, and refreshments were served prior to the meeting. Mrs. O. T. Smyth, Jr., Cleburne, gave the welcome address. Mrs. William M. Gambrell, Austin, State Auxiliary president, spoke on "Duties of Auxiliary Members," and her speech was followed by an open forum. At noon the auxiliary had luncheon with members of the district society.

After the luncheon Dr. Gambrell, president of the State Medical Association, and Mrs. Gambrell presided at an open forum for the auxiliary.

DEATHS

P. R. SANDERS

Dr. Preston Randolph Sanders, Big Spring, Texas, was found dead in his home May 16, 1950, from an undetermined cause.

Born in Blanco on November 14, 1905, Dr. Sanders was the son of Mr. and Mrs. J. E. and Annie Mae (Van Horn) Sanders. His early education was acquired in the Big Spring High School and Texas Agricultural and Mechanical College, College Station. Dr. Sanders was awarded a medical degree from the University of Texas School of Medicine, Galveston, in May, 1934, and later did postgraduate work at Washington University, St. Louis. After an internship at Hermann Hospital, Houston, Dr. Sanders began his medical career in Big Spring in 1935 and, with the exception of military service in the United States Army Medical Corps from 1940 to 1944, practiced there until his death. His specialty was pediatrics. About a year prior to his death he founded the Medical Arts Clinic Hospital, Big Spring, with his brother, Dr. Virgil Sanders, and sister-in-law, Dr. Nell White Sanders.

Throughout his professional life in Texas Dr. Sanders was a member of the American Medical Association and State Medical Association through Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Medical Society. He was a member of the Second District Medical Society. Dr. Sanders was a member of the Masonic Order, the Woodmen of the World, and the Methodist Church; he also belonged to the American Business Club.

In May, 1933, in Galveston, Dr. Sanders married Miss Dorothy Crume, who survives. Also surviving are a son, Scott Sanders; a daughter, Susan Sanders; his parents, Mr. and Mrs. J. E. Sanders; three brothers, Dr. J. V. Sanders and Garland Sanders, Big Spring; and Capt. Clifton Sanders, Long Beach, Calif.; and two sisters, Mrs. Gladys Carpenter, Big Spring; and Mrs. Warren Smith, Los Angeles.

W. I. WATERS

Dr. William Isaac Waters, Midland, Texas, died June 26, 1950, in Midland after an automobile accident near Garden City which also claimed the life of the physician who was to have been his associate, Dr. Abner Mackey, an orthopedic specialist formerly of Oklahoma City, Okla.

Dr. Waters was born August 11, 1912, in Coshocton County, Ohio, the son of John and Laura Waters. He earned a bachelor of arts degree from Ohio State University, Columbus, in 1937 and the same year was awarded a degree from the university's college of medicine. Dr. Waters served an internship at Union Memorial Hospital, Baltimore, and a residency at Augustana Hospital, Chicago, where he did postgraduate work with Dr. Nelson Percy. The latter months of 1940 Dr. Waters spent in Dayton, Ohio, working and studying in the field of chest diseases. From 1941 to 1946 Dr. Waters was on active duty with the United States Army Medical Corps, serving with the rank of major. He was a Japanese prisoner of war for three and one-half years and was a survivor of the Bataan Death March. After his discharge he was an assistant in surgery in the Medical Department of Tulane University of Louisiana, New Orleans, and was on the visiting surgery staff of Charity Hospital. He was the senior surgical resident at the Veterans Administration Hospital, New Orleans, from 1947 to 1948. Recently he had been in practice in Midland and Seminole and he was to have been the chief surgeon of the new Midland Memorial Hospital.

Dr. Waters was a member and a fellow of the American Medical Association and a member of the State Medical Association through Ector - Midland - Martin - Howard - Andrews - Glasscock Counties Medical Society. He was active in the Kiwanis Club and was a member of the Methodist Church.

In Guam in 1945 Dr. Waters married Mrs. Gertrude Oppenheimer, who survives him. Other survivors are his parents, Mr. and Mrs. John Waters, West Lafayette, Ohio; and a sister.

R. B. BLEDSOE

Dr. R. B. Bledsoe, Lufkin, Texas, died after an extended illness May 4, 1950, in a Lufkin hospital.

Dr. Bledsoe was born April 19, 1870, in Houghton, La. At the age of 16 he came to Texas with his family and attended the public schools in Hardin County. He practiced in Rockland and Matt a short while prior to his graduation in 1902 from the Atlanta College of Physicians and Surgeons, now known as Emory University, Atlanta. Dr. Bledsoe moved to Lufkin in 1902 and practiced there until forced by ill health to retire several years prior to his death.

Throughout his professional career in Texas Dr. Bledsoe was active in the State Medical Association and the American Medical Association. One of the eight charter members of Angelina County Medical Society, he served as its first secretary-treasurer in 1904. In 1930-1931 he was the first vice-president of the State Medical Association and in 1938 was elected to honorary membership in the Association. Dr. Bledsoe helped to establish Angelina County Hospital. Formerly he was county and city health officer and a member of the school board, city council, and chamber of commerce. For many years he was a deacon of the First Baptist Church, Lufkin, and a member of the Masonic Lodge.

Dr. Bledsoe is survived by his wife, a brother, Tom Bledsoe, and a sister, Mrs. J. W. Lewis, all of Lufkin.

A. G. SNEED

Dr. Arch Gustavus Sneed died of coronary thrombosis at his home in Denison, Texas, on May 13, 1950.

A native of Conway, Ark., Dr. Sneed was born March 9, 1891, the son of U. D. and Emma (Jordan) Sneed. As a small boy he moved with his parents to Grayson County, Texas, where he received his early education at Dorchester and Pottsboro. He received a bachelor of arts degree from Austin College, Sherman, in 1916, and a doctor of medicine degree from Baylor University College of Medicine, Dallas, in 1921. After serving an internship at Baylor Hospital, Dallas, Dr. Sneed became house physician at the Missouri-Kansas-Texas Hospital, Denison. He remained in Denison as local surgeon for the Missouri-Kansas-Texas Railway and as a private practitioner in internal medicine and surgery until 1945, when a heart attack compelled him to give up active practice. He had served on the medical advisory board of the Veterans Administration in Dallas since 1945 but had maintained his residence in Denison.

Dr. Sneed was a member of the American Medical Association and State Medical Association through Grayson County Medical Society, which he had served as president. He was elected to honorary membership in the state organization in 1949. He was a deacon in the First Baptist Church of Denison, a thirty-second degree Mason, and a

member of the Scottish Rite and Shrine. He was a past president of the Denison Lions Club and a director of the State National Bank. Dr. Sneed had served at one time with the U. S. Navy.

Dr. Sneed's wife, the former Miss Lorine Monzingo, whom he married in Magnolia, Ark., in 1927, died in 1944. He is survived by two sons, Edmund David Sneed and Richard Floyd Sneed; three brothers, Harvey Sneed, Lubbock; Carl Sneed, Sherman; and Fred Sneed, Denison; and one sister, Mrs. J. W. McMennamy, Denison.

I. H. SHOSS

Dr. Isadore Hyman Shoss, Houston, Texas, died July 8, 1950, in a local hospital after a long illness.

Dr. Shoss was born February 11, 1921, in Houston, the son of Max and Rebecca Shoss. He received his early education in Sam Houston High School, Houston, and the University of Texas, Austin. On July 16, 1945, Dr. Shoss was graduated from Baylor University College of Medicine, Houston, afterward serving an internship in Jefferson Davis Hospital, Houston. He began his practice in 1946 in Houston, where he had a medical, surgical, and dental clinic, and was practicing in Houston at his death.

A member of the American Medical Association and State Medical Association through Harris County Medical Society, Dr. Shoss was president of Phi Delta Epsilon medical fraternity at Baylor University College of Medicine from 1944 to 1945. He was a member of the M. M. Society.

DR. I. H. SHOSS

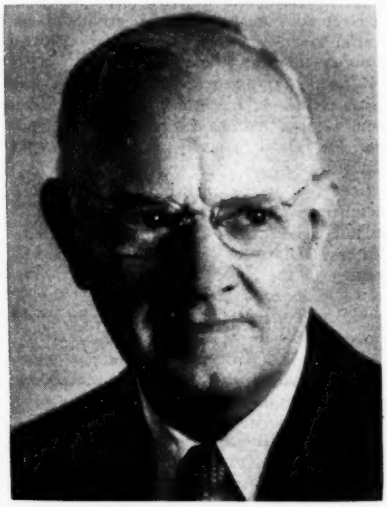
On November 12, 1944, in Fort Worth, Dr. Shoss married Miss Beverly Mallin, who survives, as do their daughter, Sandra Michelle Shoss; his parents, Mr. and Mrs. Max Shoss, Houston; and three brothers, Dr. Milton Shoss, Cape Girardeau, Mo.; Dr. Joseph Shoss, St. Louis; and Samuel Shoss, Houston.

W. M. STROZIER

Dr. William Marcel Strozier, Houston, Texas, died in a Houston hospital June 26, 1950, from cerebral thrombosis after suffering a stroke last December.

Dr. Strozier was born July 4, 1880, in Willis, Texas, the son of Augustus B. and Emily (Fisher) Strozier. He was the brother of Dr. A. B. Strozier, Coleman. Dr. Edgar Strozier, Houston; and the late Dr. Frank Strozier, Houston. After receiving his preliminary education in the private and public schools of Willis and the Willis Male and Female College, Dr. Strozier was graduated in 1906 from the University of Texas School of Medicine, Galveston. He began his medical career in Santa Anna, where he remained from 1906 to 1920, then moved to Houston, where he practiced until illness caused his retirement in 1949. His specialty was diseases of the eye, ear, nose, and throat.

A member continuously from 1909 until 1949 of the State Medical Association and the American Medical Association, Dr. Strozier was a member successively of Coleman County and Harris County Medical Societies. He was a former president and secretary of the Coleman County society. Dr. Strozier was also a member of the Ophthalmological and Otolaryngological Section of the Houston Academy of Medicine, formerly serving as its president. He was an assistant professor of clinical ophthalmology at Baylor University College of Medicine. He served on the active staff of several charity hospitals in Houston. A Methodist, Dr. Strozier was a member of the Masonic Order.



DR. W. M. STROZIER

Dr. Strozier married Miss Mary Enloe on January 1, 1907, in Sour Lake; she survives as do his son, Dr. William Enloe Strozier, Beaumont; two daughters, Mrs. Keith Stewart, Conroe, and Mrs. Tom Lewis, Houston; two brothers, Dr. A. B. Strozier, Coleman, and Dr. Edgar Strozier, Houston; two sisters, Mrs. Harry L. Washburn and Mrs. C. L. Matthews, Houston; and nine grandchildren.

E. W. BERTNER

Dr. Ernst William Bertner, seventy-second president and former trustee of the State Medical Association of Texas, died July 28, 1950, in Houston, Texas, of cancer.

The son of Gustave and Anna (Miller) Bertner, Dr. Bertner was born August 18, 1889, in Colorado City, Texas, where he received his early education. He was graduated from the New Mexico Military Institute, Roswell, and obtained his medical education at the University of Texas Medical Branch, Galveston, from which he was graduated in 1911. He did hospital work at the Willard Parker Hospital, Saint Vincent's Hospital, and the Manhattan Maternity Hospital in New York before opening his practice in Houston. In May, 1917, Dr. Bertner was commissioned in the

Medical Corps of the United States Army and was assigned to duty with the British Army. After serving overseas with the British for several months, he was transferred to the American Expeditionary Force and was assigned to duty in France. He was discharged in June, 1919, with the rank of major and returned to Houston to resume practice. In 1921 he went to Baltimore for a year of postgraduate study in surgery, gynecology, and urology at Johns Hopkins Hospital, and upon his return to Houston he limited his practice to surgery and gynecology.

Throughout his professional career Dr. Bertner was active in medical organizations. He served as president of Harris County Medical Society, the Post Graduate Medical Assembly of South Texas, the Texas Surgical Society, the Texas Association of Obstetricians and Gynecologists, and, as previously mentioned, the State Medical Association. He was a member of the executive committee of the Central Association of Obstetricians and Gynecologists, state counselor and fellow of the American College of Surgeons, member of the executive committee of the Texas Social Hygiene Association, and

DR. E. W. BERTNER

vice-president of the American Cancer Society and chairman of the executive committee of its Texas Division. He was a member and fellow of the American Medical Association and a member of the Southern Medical Association, Texas Railway and Traumatic Surgical Association, American Urological Association, and American Gynecological Association. He had served as vice-chairman of the Houston Board of Health. Dr. Bertner was former chief of staff of Jefferson Davis Hospital and Hermann Hospital and a member of the surgical staff of Memorial Hospital and Southern Pacific Hospital.

In recent years Dr. Bertner has done much in the cause of cancer research, prevention, and treatment and in the development of Houston as a medical center. In addition to participating actively in the state and national cancer societies, he served as acting director of the M. D. Anderson Hospital for Cancer Research during its first four years. He was instrumental in the founding of the Texas Medical Center and had served as its president since its establishment in 1945. He was professor and chairman of the Department of Gynecology at Baylor University College of Medicine and lecturer in gynecology and oncology at the University of Texas Medical Branch. In 1949 the American Cancer Society presented him an award for distinguished service in cancer research, and Jesse H. Jones fellowships totalling $25,000 have been given in his honor to the M. D. Anderson Hospital by Houston Endowment, Inc. One of the buildings at the Ochsner Institute in New Orleans has been named for Dr. Bertner in recognition of his work in the cancer field, and a street in the Texas Medical Center in Houston bears his name. In June this year an honorary degree of doctor of laws was awarded to him by Baylor University.

Dr. Bertner was a member of the Presbyterian Church. He was active in the Masonic Order, being a thirty-second degree Mason, Knight Templar, and member of the Shrine. He was recently made a knight commander of the court of honor of the Scottish Rite. He was a member of the Houston Club and the Houston Country Club.

Surviving Dr. Bertner are his wife, the former Miss Julia Williams, whom he married in 1922 in St. Louis, and two sisters, Mrs. Howard A. Tune, Little Rock, Ark., and Mrs. Ann Bertner Sheedy, New York.

S. W. BAILEY

Dr. Stanfill William Bailey, Abilene, Texas, died in an Abilene hospital July 15, 1950, from myocardial infarction.

Born August 21, 1906, in Bertram, he was the son of C. E. Bailey, D.D.S., and Mrs. Bailey. He attended Wichita Falls High School and Wichita Falls Junior College and on June 1, 1931, was awarded a medical degree from Baylor University College of Medicine, Dallas. After a year's internship at St. Paul's Hospital, Dallas, he practiced for seven years in Lueders before moving in 1938 to Abilene. There, with the exception of military service from May, 1942, to November,

DR. STANFILL W. BAILEY

1945, he practiced until shortly before his death. Dr. Bailey during his twenty-two months overseas served in New Guinea, Leyte, and the Philippines, and he was awarded the Purple Heart and Bronze Star medals.

Dr. Bailey was a member of the State Medical Association and the American Medical Association through Taylor-Jones Counties Medical Society, formerly having served the society as secretary. He was also a member of the Thirteenth District Medical Society. Dr. Bailey was chairman of the medical staff of St. Ann's Hospital for 1949. He was formerly secretary of the Hendrick's Memorial Hospital staff and was serving on its executive committee at the time of his death. A member of Phi Chi medical fraternity, he belonged to the

Exchange Club and the Boy Scout Fathers. Dr. Bailey was a steward of the First Methodist Church, Abilene.

In Dallas, on September 7, 1930, Dr. Bailey married Miss Audra Faye Darby, who survives. Other survivors are a son, Phillip Stanfill Bailey, Abilene; his parents, Dr. and Mrs. C. E. Bailey, Wichita Falls; and a brother, J. O. Bailey, D.D.S., Wichita Falls.

E. A. WRIGHT

Dr. Elva Anis Wright, Houston, Texas, died at her home July 18, 1950, of a ruptured aneurysm.

Dr. Wright was born March 20, 1868, in Hillsdale, Pa., the daughter of Jefferson and Nancy Jane (Dunlap) Wright. She was a graduate of Purchase Line Academy, Indiana County, Pa.; of Valparaiso University, Valparaiso, Ind., from which she received a bachelor of science degree in 1894; and of Northwestern University Medical School, Chicago, from which she received a doctor of medicine degree in 1900. She had taken postgraduate work at the University of Edinburgh, Scotland, and at clinics in Vienna and Berlin. She served an internship at Mary Thompson Hospital, Chicago; taught embryology at Northwestern University for one year; and was in practice at Lake Forest, Ill., for ten years before moving to Houston, where she was active in the practice of obstetrics and gynecology until her death. She was emeritus professor of obstetrics at Baylor University College of Medicine.

From the time of her arrival in Houston, Dr. Wright was a member of the Harris County Medical Society, State Medical Association, and American Medical Asociation. She had been an honorary member of the State Medical Association since 1941. She was a charter member of the Post Graduate Medical Assembly of South Texas, a past president and president emeritus of the Texas Tuberculosis Association, a past president of the Southern Tuberculosis Conference, and the founder and president since its organization of the Houston Anti-Tuberculosis League. She established the first clinic for children at the city health department in Houston, and for many years was the examining physician for the Harris County School of Girls and the juvenile court for girls. Dr. Wright was a founder and charter member of the Houston Altrusa Club, a life member of the Save-a-Life League, a member of the Woman's Forum and Woman's City Club, and a member of the Chamber of Commerce health committee. She announced plans in 1949 to set up a scholarship for research to help fight tuberculosis, and prior to her death requested that friends who wanted to send flowers, instead contribute to the Elva Wright Memorial Fund to assist students specializing in the study of tuberculosis. On June 20, 1950, Dr. Wright was awarded a gold certificate for fifty years' service as a doctor.

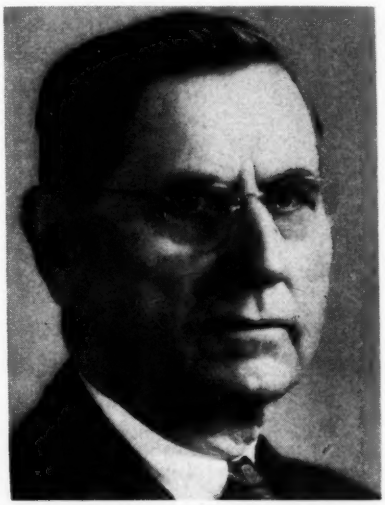
DR. ELVA A. WRIGHT

Surviving Dr. Wright are two brothers, John Wright, Hillsdale, Pa., and Blair Wright, Punxsutawney, Pa.; a sister, Mrs. J. S. Bunch, Oswego, Ill.; and two nephews, Roy Wright, West Columbia, and Merrill C. Wright, Houston.

A. C. CORRY

Dr. Albert Clarence Corry, Farmersville, Texas, died August 15, 1950, in a McKinney hospital from coronary thrombosis.

Born in Lisbon, La., on November 19, 1875, Dr. Corry was the son of A. A. and Elizabeth (McClendon) Corry. He attended public schools in Rockwall, the University of Texas School of Medicine, Galveston, and Tulane University of Louisiana, New Orleans, La. In 1902 he was graduated from the Atlanta College of Physicians and Surgeons, now known as Emory University, Atlanta, Ga. Dr. Corry practiced in Powderly, Texas, for one year, in Copeville for fifteen years, and in Farmersville for twenty-eight years. He was a member of the staff of the McKinney City Hospital and a local surgeon for the Santa Fe Railway Company.



DR. ALBERT C. CORRY

A member throughout his professional career of the American Medical Association and the State Medical Association, Dr. Corry in 1937 and 1944 served as president of the Collin County Medical Society. He was a member of the Fourteenth District Medical Society. A Methodist and a Mason, he was a director and a member of the board of stewards of the First Methodist Church. Dr. Corry was a Boy Scout director, physician for the local football team, and a past president of Rotary Club.

In 1908 Dr. Corry married Miss Margie Yeary, who survives. Also surviving are a daughter, Mrs. John McCraw, Farmersville; four brothers, Dr. J. Frank Corry, Rockwall, E. H. Corry, Joshua, Gus Corry, Nevada, and Charles Corry, Fort Worth; a sister, Mrs. Angie Corry Harris, Irving; and two grandchildren.